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A TEXT-BOOK OF MEDICINE

FOR STUDENTS AND PRACTITIONERS

VOLUME II

A TEXT-BOOK OF MEDICINE

FOR STUDENTS AND PRACTITIONERS

BY

DR. ADOLF v. STRÜMPELL

PROFESSOR OF SPECIAL PATHOLOGY AND THERAPEUTICS AT THE
UNIVERSITY OF LEIPSIC

Fourth American Edition

*TRANSLATED BY PERMISSION FROM THE SEVENTEENTH
REVISED GERMAN EDITION*

*WITH EDITORIAL NOTES, ADDITIONAL CHAPTERS, AND
A SECTION ON MENTAL DISEASES, BY*

HERMAN F. VICKERY, A.B., M.D.

INSTRUCTOR IN CLINICAL MEDICINE, HARVARD UNIVERSITY; VISITING PHYSICIAN TO THE
MASSACHUSETTS GENERAL HOSPITAL; MEMBER OF THE ASSOCIATION OF AMERICAN
PHYSICIANS; FELLOW OF THE MASSACHUSETTS MEDICAL SOCIETY, ETC.

AND

PHILIP COOMBS KNAPP, A.M., M.D.

EX-PRESIDENT OF THE AMERICAN NEUROLOGICAL ASSOCIATION, OF THE NEW ENGLAND
SOCIETY OF PSYCHIATRY, AND OF THE BOSTON SOCIETY FOR PSYCHIATRY AND NEUROLOGY; CLINICAL INSTRUCTOR IN DISEASES OF THE NERVOUS SYSTEM, HARVARD
UNIVERSITY; PHYSICIAN FOR DISEASES OF THE NERVOUS SYSTEM, BOSTON CITY HOSPITAL; FELLOW OF THE ROYAL SOCIETY OF MEDICINE, OF THE MASSACHUSETTS MEDICAL SOCIETY, ETC.

*WITH SIX PLATES, THREE OF WHICH ARE IN COLOR, AND TWO
HUNDRED AND TWENTY-FOUR ILLUSTRATIONS IN THE TEXT*

VOLUME II

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A TEXT-BOOK OF MEDICINE

VOLUME II

VI. DISEASES OF THE ORGANS OF LOCOMOTION

CHAPTER I

ACUTE ARTICULAR RHEUMATISM

Ætiology.—Acute articular rheumatism is an infectious disease. This is shown by all its clinical and anatomical characteristics, and although we are not yet definitely acquainted with the specific organized poison which gives rise to it (*vide infra*), still, it is impossible to come to a correct understanding of the symptoms and the course of the disease, unless we assume that it is caused by an infectious germ.

Like many other infectious diseases, acute articular rheumatism is often indisputably endemic and epidemic. According to Hirsch, the disease is most prevalent in the temperate zones, being much rarer in cold and tropical latitudes; but even in Europe it is by no means uniform in its frequency, and certain districts of England, Belgium, and Russia are said to be almost exempt from it. It is also possible to observe epidemic influences with regard to the frequency of its appearance, as already intimated. In Leipsic, where articular rheumatism is one of the most frequent of acute diseases, we observed for years that at certain times there were only a few cases, while at others there was a striking increase in their number. We have made the same observation in Erlangen and in Breslau. Usually attacks are most prevalent in the winter and spring months, but again it is sometimes in summer that the disease is especially common.

Among the exciting causes of the disease, taking cold is always mentioned as of first importance; and, in fact, it cannot be denied that the influence of cold does often seem to contribute to the occurrence of the disease. This result, however, seldom follows a single severe exposure, but it much more frequently follows long-continued "rheumatogenous influences," as we may call them, and in particular the long-continued influence of wet and cold, certain occupations—for example, washing and scrubbing—inhabiting unhealthy damp dwellings, and the like. Those who follow certain callings are especially subject to articular rheumatism; thus, servant girls and coachmen are frequently victims to the disease. And yet it is possible to regard all these injurious influences as being merely indirect causes, assuming that they favor the development or action of the specific microörganisms; and, furthermore, it is by no means exceptional to see a case of articular rheumatism in which no history of exposure to cold can be obtained.

Sex exerts no special influence upon the frequency of the disease. As to age, acute articular rheumatism is most frequent in young adults between fifteen and thirty-five years old. In later life, and particularly in old age, it is much rarer. In children six years old or more the disease is not especially infrequent, but in younger children it occurs only exceptionally. We may be permitted to mention a single interesting case which we met with in Leipsic, where a child who died when only a few days old, and whose mother at the time of its birth was suffering from a severe attack of acute articular rheumatism, was found to have multiple purulent arthritis.

Of precisely what variety the special germ of acute rheumatism may be we do not yet know. Some investigators incline to the view that acute articular rheumatism is a staphylococcus infection, and therefore not a specific disease, but a special form of mild septic infection. Others, however, claim to have found a specific germ of infection in the blood and in the exudation of the inflamed joints. But none of these assertions has been confirmed. It is indisputable that a close clinical affinity exists between acute articular rheumatism and septic diseases (in a broad sense of the term). As is well known, multiple swellings of the joints are one of the most frequent symptoms of septic infections, and the appearance of endocarditis or of an inflammation of other serous membranes (pleurisy, pericarditis, etc.) is fully as characteristic of all staphylococcus and streptococcus infections as it is of polyarthritis. If, however, we are not yet inclined to accept the designation of acute articular rheumatism as a mild septic infection, then it would seem advisable to speak of all the forms of disease which belong in this class and which are closely related to one another, as due to a "rheumatic infection," employing the word rheumatic with a special idea of ætiology.

If we conceive of articular rheumatism in the way just indicated as a clinically well-characterized constitutional infection—perhaps with staphylococci—the next question is the place of infection, and its gate of entrance. It seems probable that the place is not the same in every case. Very often it is impossible to determine the path by which infection entered; yet we may sometimes ascertain by careful questioning that the arthritis was preceded by a mild sore throat, or a laryngitis, or an intestinal disturbance. Indeed, we often find even small abrasions of the skin, paronychia, and similar lesions. It is probable that such statements of the patient and discoveries of the physician indicate the starting point of the infection in the individual case. Not infrequently, the organs of the throat, particularly, appear to be the points of entry for the infection (tonsils and pharyngeal tonsils).

As far as the cases of so-called secondary rheumatism which occur subsequently to scarlet fever, epidemic meningitis, and other acute diseases are concerned, it is probable that, in some instances, these are due to a secondary rheumatic infection, although, on the other hand, it is also possible that the special germs of these infectious diseases may produce inflammation of the joints—e. g., multiple purulent arthritis is probably due in most cases to streptococci. The appearance of acute articular rheumatism in a case of already existing valvular disease is of practical importance, and very frequently this process is to be regarded merely as a fresh acute infection, starting from the old focus.

A very noteworthy fact is that acute articular rheumatism cannot be numbered among those infectious diseases which usually occur but once in the same individual. On the contrary, the disease has the same peculiarity as pneumonia and erysipelas, of appearing with noticeable frequency several times in the same individual, so that when a person has once had articular rheumatism, even though it has left no sequelæ behind it, he seems to be more liable than before to the disease. It is not exceptional to meet persons who have had five or more attacks of acute articular rheumatism.

Symptomatology.—The chief symptom of acute articular rheumatism is an acute febrile synovitis, which almost always affects several joints. The synovitis is associated with the usual local phenomena of swelling and tenderness in the parts affected. Often this articular affection is the first symptom, and, indeed, it may be the only symptom of the disease. It is, however, by no means exceptional for the arthritic trouble to be preceded by certain prodromal or initiatory symptoms, as is true of other infectious diseases. These prodromata consist either of a slight general malaise, or of certain local symptoms. It is not rare, as already mentioned, to have sore throat, or, as we have repeatedly had opportunity to observe, laryngitis. Still, the precursors are usually mild; and they may, as we have said, fail to appear at all, or at any rate they may easily escape notice. In some few cases the only symptoms at first are febrile; there may be no other marked constitutional symptoms, or there may be a typhoidal condition with enlarged spleen, and diarrhea. Then, some days later, the characteristic swelling of the joints appears (Wagner). Taken in connection with the view of the nature of articular rheumatism which we have elaborated above, this behavior has nothing inexplicable about it—merely, the constitutional symptoms develop some little time before the local manifestations of the disease.

The articular disturbance is almost always very rapid in its development. Some of the larger joints are usually first affected, and perhaps those of the lower extremities somewhat oftener than those of the upper. It is extremely exceptional for all the joints that are affected to be attacked at one and the same time. It is somewhat characteristic of acute articular rheumatism that it "jumps from one joint to another." To-day this joint will be affected and to-morrow that, while the joint first attacked may still remain diseased or undergo rapid recovery. Thus, there may be either a few joints affected or many, in varying sequence, and they may sometimes be affected rapidly and at other times more slowly. In many of the milder cases the disease is an extremely temporary one, while in others it may attach itself most persistently to some one or more joints.

There is usually fever in addition to the arthritis, but it is not usually very high, seldom exceeding 103° F. (39.5° C.). The fever, on the whole, corresponds with the arthritic phenomena, and does not present an especially typical curve, but one which is irregularly remittent. We have scarcely ever seen the disease begin with an initial rigor, and the so-called "general febrile symptoms" of headache, stupor, and subjective feeling of heat are, as a rule, ill marked in acute rheumatism. This indicates that the constitutional infection does not, owing to the peculiar nature of rheumatic infection, attain great severity. The skin is noticeably inclined to perspiration, but the perspiration is not at all a result of any sudden fall in temperature such as is seen in other diseases.

The course of the disease is marked by alternate ameliorations and aggravations of the local symptoms and of the fever, and lasts, particularly if it is not treated, one or more weeks, or a still longer period. Often the entire course of the disease under such circumstances is made up of separate "attacks" (*vide infra*), there being repeated improvement followed by articular swelling and fever. At last, however, the symptoms entirely disappear, and the patient enters upon a tedious but complete convalescence.

In contrast with this simple course the clinical picture may be, in other cases, much more complex, for the peculiar infectious character of articular rheumatism renders possible many complications and peculiarities. The protean character of the disease will be evident upon a perusal of the following description of the symptoms referable to the different organs of the body.

SYMPTOMS REFERABLE TO THE DIFFERENT ORGANS, AND PECULIARITIES IN THE COURSE OF THE DISEASE. 1. *Joints and Sheaths of the Tendons.*—The favorable termination of most cases of acute articular rheumatism prevents us from often examining the anatomical changes in the affected joints, but there can be no doubt that in most instances the trouble is merely a simple serous synovitis—that is, an inflammation of the synovial membrane, with an exudation into the cavity of the joint, composed mainly of serum with but little admixture of fibrin and pus. The synovial membrane itself, in the cases which do come to autopsy, is usually very little affected. It is somewhat injected, opaque, and thickened. Necrosis of the cartilages is seen only in severe cases, or in those which have lasted a rather long time. From a clinical standpoint the articular disturbance is noticeable chiefly for the pain which it causes the patient upon every movement of the joint and any pressure upon it. The tenderness is often in striking contrast with the slight objective signs of inflammation, for a joint which is extremely sensitive may appear to be scarcely at all diseased. Usually, however, the joints exhibit the signs of synovitis. The effusion into the joint produces an evident swelling, which can be seen particularly well in the knees, but also in the joints of the ankle, wrist, shoulder, and elbow, and sometimes even in the smaller joints of the fingers and toes, particularly the great toe.

It is rather exceptional to detect swelling of the hip joint. It should be remembered, however, that the swelling in the region of the joint, particularly the ankle or wrist, is often less the result of a synovial effusion than of an inflammatory periarticular œdema. This œdema, for example, may extend over almost the whole posterior surface of the hand. The joints are by no means invariably the only parts attacked. Not infrequently there are analogous inflammatory changes visible in the sheaths of the tendons, the bursæ, and perhaps, in many cases, even the fasciæ and muscles. The skin over the affected joints often has an inflammatory blush, which is usually pale red and spotted, and can be best seen at the ankle, knee, and wrist. It has been maintained that the cutaneous sensibility is diminished over the joints affected, but we regard this as a mistake.

As might be expected, the number of joints attacked and the sequence in which they are attacked differ greatly in different cases; but almost invariably a number of joints suffer, so that any monarticular arthritis should not be regarded as rheumatic except after careful deliberation (*vide infra*, diagnosis). It should be said that, in mild cases, there may be only two or three joints affected, these being usually some of the larger joints of the extremities; and of these, one may be so much worse than the others that their participation in the trouble can be ascertained only by careful questioning and examination. In severe cases, on the other hand, the number of joints attacked is often very great. Such patients become extremely helpless, because any movement is possible, if at all, only under the penalty of very severe suffering. The patient usually lies with bended knees and feet curved so as to be concave on the plantar surface, and screams with pain at any attempt to change his position. The joints of the trunk sometimes participate in the disease, but hardly ever except in the severe cases; the articulations of the vertebræ, the sterno-clavicular joint, the articulation of the lower jaw, and the symphysis pubis, are then particularly apt to be affected.

The fugitive character of the arthritis has been spoken of as characteristic of acute articular rheumatism, and, indeed, it is not infrequently the case that comparatively large swellings of the joints soon abate and yield to new disturbances in other joints; but, on the other hand, the disease may persist very obstinately in a single joint. In this case one joint or, rarely, several joints are attacked with marked severity, either from the start or subsequently to milder affections of other joints, and often remain for weeks swollen or painful, long after all other symptoms have vanished.

2. *Cardiac Symptoms.*—The condition of the heart in acute articular rheumatism is next in importance to that of the joints. The physician should therefore, in every case, even the mildest, maintain a continuous watch over this organ. In 1836 Bouillaud made careful auscultatory investigations in this disease, and was thus the first to discover that the course of acute articular rheumatism is, with noticeable frequency, accompanied by endocarditis, and sometimes even by pericarditis. Complications of this sort may occur in any case, whether mild or severe, or may be absent in any case, even the worst. They may develop at the beginning or later on in the course of the disease.

Their development is often unattended by any subjective symptoms, so that they can be recognized only by careful physical examination. In many cases, however, the onset of cardiac disease is marked by a fresh exacerbation of the fever, or possibly by palpitation, or by painful sensations in the præcordia, or by dyspnœa.

We will consider first rheumatic endocarditis. This is almost always the benign verrucous variety (see Vol. I). It is far more prone to attack the mitral than the aortic valve, and is accordingly usually betrayed by a blowing systolic murmur at the heart's apex. Uncertainty may be cast upon the diagnosis by the fact that functional murmurs are not very infrequent at the apex or at the base of the heart in cases of acute articular rheumatism. We once observed a case of "hyperpyretic rheumatism" (*vide infra*) in which there was an evident murmur of this sort during life, and yet at the autopsy we were able to assure ourselves of the normal condition of the cardiac valves. Even an expert may for a time be in doubt as to the significance of many cardiac murmurs, and this explains in part the conflicting statements as to the frequency of cardiac complications in acute rheumatism. In general, one may say that such complications occur in twenty-five to thirty-three per cent of the cases. The ultimate results of this endocarditis we do not need to describe over again in this connection (see the chapters on Acute and Chronic Endocarditis). Complete recovery is possible. Often, however, the lesion gives rise to a chronic endocarditis—that is, to a cardiac valvular disease which lasts through life.

The close connection between endocarditis and the arthritis must formerly have seemed very puzzling, despite the many hypotheses made to explain it. Our present conception of articular rheumatism as a general infectious disease renders the frequent development of endocarditis easily explicable. It is due to a settlement of the germs, which are circulating in the blood, upon the cardiac valves. As a matter of fact, staphylococci are almost invariably found in the inflammatory deposits of " rheumatic " endocarditis. The endocarditis, therefore, is not strictly a " complication," but a symptom of the disease.

Rheumatic pericarditis is not infrequent, although less common than endocarditis. The only certain way to recognize it is by a characteristic friction sound; and even when this is heard there may be a doubt as to its significance, inasmuch as functional murmurs are not infrequently heard at the base of the heart. The pericarditis is of serofibrinous nature. Sometimes it is of slight severity, but it may be extremely severe, with a large effusion and the most urgent dyspnœa. In rare instances this pericarditis proves fatal. Usually, however, recovery ensues, although in severe cases there may be obliteration of the paricardial sac, with its possible consequences.

As to the way in which pericarditis develops, it is probable that in most cases there is a direct infection of the pericardium from the blood. In some cases, however, we are inclined to assume that the infection of the pericardium comes from the endocardium, and probably in most instances from the aortic valves. We have repeatedly found a distinct aortic insufficiency to have been left behind after rheumatic pericarditis, the heart having previously been healthy. Besides rheumatic endocarditis and pericarditis, it is positive that rheumatic myocarditis also appears at times. We are especially justified in assuming its presence when permanent irregularity of the pulse occurs without a murmur. Continued acceleration of the pulse or bradycardia may be associated with myocarditis. But these may also be caused by simple toxic functional disturbances. Subjective disturbances need not necessarily be united with myocarditis. At times, however, abnormal sensations in the heart may arise (palpitation, stenocardia). Myocarditis may disappear without leaving any demonstrable consequences, or it may be the basis for a subsequent chronic myocarditis.

We should also mention that there may be functional cardiac derangement without any grave anatomical lesion. We have already spoken of the functional murmurs. There may also be a rapid and irregular pulse, and, in rare instances, attacks of angina pectoris of apparently purely nervous origin.

3. *Serous and Mucous Membranes.*—We have not only pericarditis in association with articular rheumatism, but also pleurisy and peritonitis, so that we may say that in acute articular rheumatism the incidence of the disease is mainly upon all serous membranes, inclusive of the joints. It should be said that rheumatic pleurisy, and particularly rheumatic peritonitis, are much less frequent than endocarditis and pericarditis, but we have repeatedly seen in severe cases rheumatic pleurisy, with serous effusion, on one or even on both sides. In such severe attacks it is not exceptional to find several serous cavities simultaneously involved—for instance, the pericardium and pleura; so that in such instances the inflammation may have extended directly from

the pericardium to the pleura, or *vice versa,* although direct infection of the pleura by the blood is perfectly possible. Rheumatic peritonitis, indicated by distention of the abdomen with a moderate exudation and by tenderness, has been absolutely diagnosticated in a few cases. Here, also, the inflammation may appear independently, or may be due to extension from the pleura through the diaphragm.

The mucous membranes are seldom greatly affected in acute articular rheumatism. As we have already mentioned, in the beginning of the disease there may be catarrhal affections of the pharynx, larynx, stomach, or intestines, which lesions probably facilitate the "rheumatic" infection. Bronchitis is spoken of by the older authors as frequent, but it is probably in many cases not due directly to the rheumatism, but is a complication, just as in any disease attended by great prostration.

4. *Skin.*—Cutaneous phenomena are not infrequent in the course of acute articular rheumatism. A prominent symptom of the disease is the tendency to profuse perspiration. The perspiration often has a strongly acid odor and reaction. Many patients exhibit an abundant crop of sudamina, the back in particular being sometimes entirely covered. Sometimes there are other cutaneous eruptions. In a whole series of cases we observed erythema nodosum. This affected the lower extremities more than the upper. Urticaria is not very infrequent, while herpes labialis has been very rare in our experience. It is well known that arthritic affections and the so-called "hemorrhagic diseases" are in many ways related to each other, and it is therefore an interesting fact that extensive hemorrhagic disturbance of the skin also occurs in connection with acute articular rheumatism, as we have repeatedly had opportunity to observe. We have seen several cases of hemorrhagic urticaria; wheals appear upon the skin, and a hemorrhage takes place into their centers and spreads gradually. There may also be simple cutaneous ecchymoses. These may in severe cases be merely one symptom of a general hemorrhagic diathesis, with hemorrhages from mucous membranes. The occurrence of all these symptoms shows clearly that no sharp dividing line can well be drawn between the various disorders. They all certainly belong to the group of "rheumatic infections."

5. *The Muscles and Nervous System.*—The condition of the muscles is very important in many cases of acute polyarthritis. They are often quite painful on pressure, and apparently somewhat swollen about a joint that has been long affected. The muscular atrophies and muscular paralyses that often remain after the arthritis has disappeared are especially important.

According to a general law, there are certain definite trophic relations between a joint and the muscles belonging to it whereby almost every severe and persistent disease of the joint is necessarily followed by an atrophy of the affected muscles. The extensors of the joint are most affected by this atrophy. The causes of this muscular atrophy of arthritic origin are not yet fully explained. The explanation given by Charcot, that the cause of the atrophy is a "reflex" implication of the trophic centers in the cord due to the disturbance in the joint, seems to us hardly satisfactory. The inactivity of the muscles in consequence of the painfulness and difficulty of all movements of the joints may well be accepted as a partial cause of the resulting atrophy. But, on the other hand, it is possible that special local influences come into play

—thus, direct extension of the inflammatory process, the local effect of toxins that have developed, etc. If an attack of acute articular rheumatism affects any one joint for a long period there is a secondary atrophy of the corresponding muscles. This is seen most often, and in its most typical form, when there is obstinate trouble in the shoulder joint, the deltoid becoming extremely atrophied. This atrophy of the muscles may contribute largely to the sum total of functional derangement. We have repeatedly seen cases where the patient could hardly lift his arms at all, although the inflammation of the shoulder joint had passed away, and that without leaving any anchylosis. It is therefore entirely justifiable to speak of a rheumatic paralysis. We have seen similar muscular paralyses after acute articular rheumatism in the rest of the muscles of the upper arm; also in the quadriceps extensor, and once in the serratus magnus. It is noteworthy that the atrophied muscles respond promptly to the faradic current, and never exhibit the reaction of degeneration.

Chorea (*q. v.*) may be a sequel of acute articular rheumatism, and it is one of the nervous symptoms which may arise in connection with it. This complication is seen most frequently in children. Endocarditis may accompany it, but it does not always do so.

There are certain peculiar cases of acute articular rheumatism which excite the greatest interest. In these, very severe cerebral symptoms are developed, often most acutely. They are therefore called "cerebral rheumatism," or, as they are almost always characterized by an extraordinarily high temperature, another name is "hyperpyretic articular rheumatism." In these cases the disease may exhibit severe nervous symptoms from the start, particularly delirium; or it may at first run an apparently favorable course, and not change for the worse until after several days, or even at a later period. The change may be quite abrupt. The temperature rises to 104° or 106° F. (40° to 41° C.). There are great uneasiness, delirium, and sometimes also signs of motor irritation, such as general convulsions, or tonic spasm of the extremities, or trismus. The face grows pale and cyanotic, the pulse small and extremely rapid. With slight interruptions the temperature continues to rise, and attains 107.5° to 109.5° F. (42° to 43° C.). This great rise is most apt to occur just before death, and there may be a still further increase of temperature after death occurs. As has been implied, the termination is usually unfavorable. It is only in exceptional cases that recovery takes place.

It has been stated that cerebral rheumatism attacks mainly hard drinkers and other individuals whose nervous system has been previously impaired, but our own experience does not confirm this view. No case is absolutely secure from the occurrence of hyperpyrexia, but it is a very rare phenomenon, occurring perhaps once in several hundred cases. On post-mortem examination the brain seldom shows any change in these cases of cerebral rheumatism. We are therefore obliged to regard the condition as the result of an unusually severe infection, affecting chiefly the intellectual, motor, and thermal centers.

Cases have also been reported which presented actual anatomical lesions of the brain—in particular, purulent meningitis. Probably, however, most of these cases were falsely diagnosticated, the observer having confounded articular rheumatism with epidemic meningitis, pyæmia, and similar diseases. Of course, if there be endocarditis, cerebral embolism is possible.

Mental derangements deserve a brief mention. They rarely occur during the course of the disease, but are somewhat more frequent after it has terminated. We may have either melancholia, attended with marked excitement or anxiety, or a more general insanity. The prognosis is usually favorable.

6. *Other Viscera.*—Other parts of the body than the heart, the serous membranes, and the brain, are seldom much affected in articular rheumatism. Lobar pneumonia occurs only in especially severe cases, but in such it may attain quite a considerable development and occasion great dyspnœa. It usually requires quite a long while to recover from it. Whether we actually have to do in these cases with a specific pneumonia, or with an ordinary lobar pneumonia as a complication, has not yet been determined. It is also possible for lobular, deglutition pneumonia to develop in severe articular rheumatism. Acute nephritis has been demonstrated in some instances, but this is very rare. The spleen, as already mentioned, may be somewhat swollen; but, as a rule, we do not find the distinct splenic tumor which is present in other acute infectious diseases.

7. *General Symptoms.*—In many cases the general condition of the patient is but little affected, but in others the disease seems to exert a peculiar influence upon the constitution. This may show itself in a striking anæmia, and we have observed this repeatedly where there was no cardiac complication. Ordinarily there is a slight leucocytosis. A dangerous but extremely rare complication has already been briefly referred to—namely, the occurrence of a general hemorrhagic diathesis. This is almost always associated with high fever and great prostration, and is usually fatal. In all such cases there is probably profound sepsis.

Course, Duration, and Prognosis.—Acute articular rheumatism may be described as generally a benign disease, for it usually terminates in recovery. It is only in very few cases that an unfavorable termination takes place immediately, whether as the result of pericarditis or other severe cardiac complication, or from hyperpyrexia, or the development of a general hemorrhagic diathesis.

The entire duration of the disease varies greatly according to its severity. There are mild cases which terminate in a few days, and, on the other hand, the disease may be very tedious, lasting for weeks and months. This latter form is seen, upon careful consideration of the whole course of the disease, and particularly of the temperature curve, to be made up of separate attacks, which can often be very clearly distinguished. In each attack of this sort (see Fig. 103) the temperature rises for some days and then slowly abates, so that the entire length of an attack is about seven to thirteen days. Then there is an afebrile interval of a few days, followed by a fresh rise of temperature, etc. The recurrence of the fever is usually associated with new trouble in one or more joints, or with some additional rheumatic disorder, such as endocarditis or pleurisy, while during the afebrile period the local symptoms abate, and the patient feels almost well. Friedländer has tried to deduce a series of general laws about the course of the disease, based upon this typical behavior of articular rheumatism, but in our opinion he has gone too far in this direction, although we have ourselves frequently observed this interesting course of acute articular rheumatism, with a series of separate attacks, and regard it as very characteristic of individual cases; still, there are many other forms which the disease may assume.

Quite often the violent symptoms experienced at first disappear promptly, but only to be replaced by such milder ones as pain and stiffness of the joints—these latter persisting for a long time. It is a general rule that the severity and persistency of the case correspond with the number of joints affected; but to this rule there are numerous exceptions. The disease may persist with great obstinacy in a single joint. It need hardly be said that the duration of the disease is greatly modified by the occurrence of complications, cardiac or otherwise, and such sequelæ as muscular atrophy, anchylosis of the joints, or chorea. The most important of all sequelæ is cardiac disease, and this must always be considered in giving a prognosis; for, although the disease as such does in most instances terminate in recovery, yet too often it gives rise to a tedious and usually incurable disease of the heart. It is, however, true that complete recovery from the acute endocarditis seen in articular rheumatism is possible, but in a large majority of cases recovery is not complete, and the acute passes into a chronic endocarditis. In these cases the cardiac symptoms may be developed directly, so that the patient at once begins to complain of palpitation and shortness of breath; or he may seem to regain his health completely, and a murmur which the physician alone can detect may be the only sign of the incurable injury which the body has suffered. The patient may feel perfectly well for years after, and then at last begin to suffer from the failure of compensation. Finally, we should again call attention to the important fact that after recovery from rheumatism there may be a tendency to fresh attacks of arthritis for the rest of the patient's life.

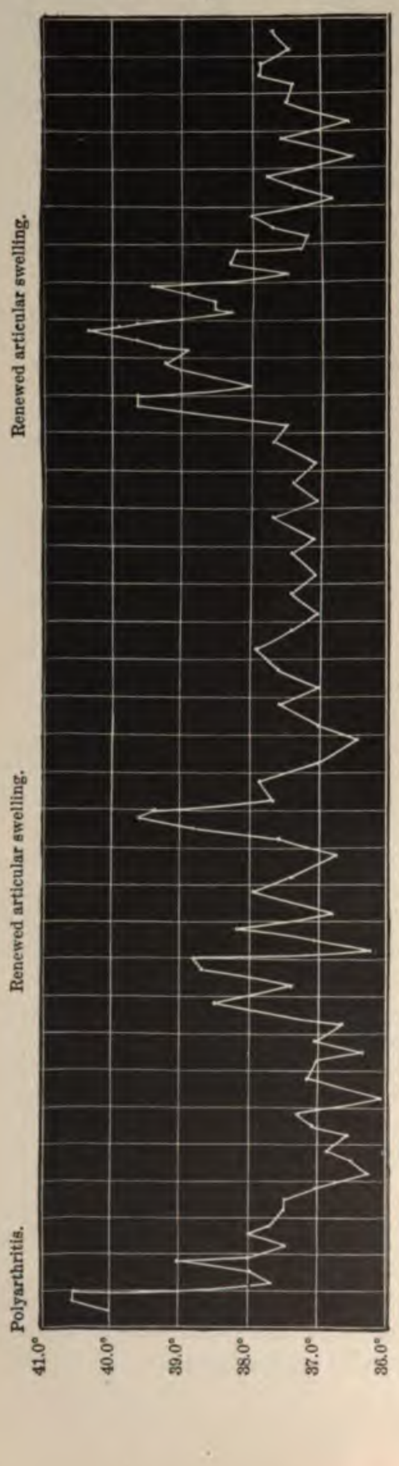

FIG. 103.—Temperature chart in acute articular rheumatism. (Erlangen Medical Clinic.)

Diagnosis.—Most cases of articular rheumatism can be easily recognized, for the acute occurrence of pain and swelling in several joints is sufficiently characteristic of the disease. It should nevertheless be borne in mind that articular swelling may also take place in the course of other diseases, and that mistakes in diagnosis are by no means impossible. When there are grave constitutional symptoms with fever from the start, we should not forget the possibility of pyæmia, or of acute osteomyelitis, since these affections occasionally give rise to the swelling of several joints. In such cases, however, careful attention to the furthest course of the disease will generally enable us to see that we cannot be dealing with a simple acute articular rheumatism. Again, after childbirth there may be swelling of the joints, of septic origin, and but distantly related to genuine rheumatism.

If a single joint be attacked, the diagnosis of articular rheumatism must be made with extreme caution. These monarticular inflammations often prove to be something entirely different—namely, fungous disease of the joint, or an osteomyelitis. The arthritis which follows gonorrhea is usually [though not invariably] monarticular (affecting especially the knee joint or wrist). In conclusion, it should be stated that it is not very rare to observe pain and swelling in various muscles and joints at the commencement of the secondary stage of syphilis, simulating an acute articular rheumatism.

Sometimes the diagnosis is doubtful in those cases which present cutaneous ecchymoses (purpura and peliosis) and erythema nodosum, because we may be unable to determine which should be regarded as the primary symptoms and which the secondary. Until we become acquainted with the specific germs which cause all these diseases, it will be merely a dispute about words, whether we shall call a particular case by one name or another. In our opinion, all these conditions have a close ætiological relation to one another.

The differential diagnosis between articular rheumatism and genuine gout is very important. We will refer to this more particularly in the discussion of gout (*vide infra*).

Finally, we must mention that acute multiple neuritis (*q. v.*), beginning with fever and severe pain in the extremities, may sometimes, if we are not sufficiently careful, be mistaken for polyarthritis.

Treatment.—Acute articular rheumatism is one of the few diseases for which we possess an undoubtedly specific and universally accepted remedy. Kolbe suggested its use, and since 1876 it has been largely employed upon the recommendation of Stricker, Buss, and others, for articular rheumatism. This remedy is salicylic acid. Although this medicine does not in all cases produce its surprisingly favorable results with equal rapidity and completeness, yet it almost invariably does produce a decided and beneficial effect upon the disease. We might even say that this influence is so constant that when salicylic acid proves entirely inefficient in a fresh case, such failure throws doubt upon the correctness of the diagnosis. Thus, where there is monarticular arthritis dependent upon some local cause, the remedy has hardly any beneficial influence. The same is true with regard to affections of the joints connected with gonorrhea, pyæmia, and similar troubles. In genuine acute articular rheumatism, on the other hand, the salicylic-acid treatment is so superior to any other that it is the first duty of the physician in every case to give this remedy a fair trial.

The two preparations of salicylic acid most used in rheumatism are the pure acid and its sodium salt, salicylate of sodium. Each of these two remedies has its peculiar advantages, but the specific influence of each is about the same. Salicylic acid should never be prescribed in solution, but always in wafers or preferably in gelodurat capsules, usually in the dose of 7.5 gr. (gm. 0.5). In this way the salicylic acid can be taken by almost any patient quite easily, especially if a little water or milk be drunk after each dose. Adults should receive a capsule every hour until about 1 or 1.5 drachms (gm. 5 to 6) have been administered. Usually there will by this time be a very decided abatement of the articular pain and swelling, while on the other hand there will also usually be such toxic "salicylic symptoms" (*vide infra*) as to forbid its further use. The salicylate of sodium is best exhibited in single large doses of 1 to 1.5 drachms (gm. 4 to 6), each dose being given with about 1 ounce (gm. 20 to 30) of peppermint water, or, still better, in wafers. The quite disagreeable taste of the medicine is only exaggerated by the addition of such substances as sirup or fluid extract of licorice, added for the sake of elegance; but the simple solution in peppermint water is quite well taken, at least by most patients. It is a very good plan to give salicylate of sodium in a glass of Hungarian wine, or in strong black coffee without sugar. The advantage of the salicylate over the pure acid consists in its being possible to give a larger dose at one time, so that it need not be taken more than two or three times a day. In general, the amount given in twenty-four hours should not exceed 2.5 drachms (gm. 10); 1.5 to 2 drachms (gm. 6 to 8) may suffice. For children the dose is, of course, smaller, about 5 gr. of salicylic acid (gm. 0.30), or, in twenty-four hours, 0.5 to 1 drachm (gm. 2 to 5) of the sodium salt.

Which of these two preparations shall be employed is, as has been stated, of little consequence. We ourselves usually prescribe, at first, capsules of salicylic acid to be taken hourly, as being most agreeable to the patient; but if our first visit be made in the evening, we prescribe a single large dose of 1 to 1.5 drachms (gm. 4 to 6) of salicylate of sodium, or aspirin (*vide infra*), so that the patient may not be disturbed every hour through the night in order to take medicine. It is often possible later on in the disease, when the patient has already acquired a distaste for the medicine, to give the salicylate of sodium as an enema. About 2.5 drachms (gm. 10) should be given, in 2 ounces (gm. 60) of water. There is no doubt that the specific effects can be obtained in this manner.

The benign influence of this remedy upon the disease is apparent in many fresh cases as early as ten to eighteen hours after treatment begins; and it is often astonishing to see how soon a patient, who before lay helpless and complaining, becomes free from pain and able to move his limbs. It must be confessed, however, that, apart from its taste, salicylic acid may produce disagreeable incidental effects. In the first place, there may be nausea with epigastric distress, and even vomiting. Tinnitus aurium may be exceedingly troublesome, and may be attended with marked vertigo. In somewhat rarer instances the mind is especially affected. Young girls in particular are often peculiarly excited; but the frame of mind is, however, in general a cheerful one. After large doses there may be an actual "salicylic delirium."

It should also be stated that respiration may be affected, becoming very deep and rapid (salicylic dyspnœa). These incidental effects have led to efforts to find substitutes for salicylic acid which shall be equally beneficial without any annoying secondary symptoms. Among the numerous substitutes that have been recommended, aspirin (acetylsalicylic acid) has thus far obtained the best reputation. At present, it is the salicylic acid preparation most frequently used in articular rheumatism. It almost invariably agrees with the patient if given in powders of 8 to 15 gr. (gm. 0.5 to 1) several times daily (ʒj to ʒjss. [gm. 4 to 6] per day), and certainly often has a favorable influence on the rheumatic affection. In addition, salol (phenylic ester of salicylic acid), salophen, and others deserve mention. But antipyretics, above all antipyrin, also pyramidon, phenacetin, lactophenin, citrophen, and many others, also affect the rheumatic process favorably, though their action is feebler than that of the salicylic preparations. These other drugs are to be employed particularly in cases of longer duration in which it becomes desirable to make repeated changes in the remedies.

Although it is not exceptional to have the rheumatic symptoms almost entirely vanish at the end of one or two days, through the use of salicylic acid, aspirin, etc., yet it is only in the minority of cases that the entire process ends with this release from pain. There is very often, sooner or later, a relapse, with fresh pain or even fresh swelling in one or more joints. It has been recommended to continue the salicylates in smaller doses for some time, in order to avert such relapses; but of late we have rather abandoned this method, for the reason that these small doses do not prevent the return of the disorder, but are calculated to give the patient a strong dislike to the remedy, and lessen his confidence in it. We therefore recommend to stop the medicine entirely as soon as the pain is wholly gone, and to guard the patient as much as possible from relapses by preventing his catching cold (*vide infra*). If there be fresh pain, we should at once resume the acid, aspirin, or a similar drug, in large doses, and thus we shall very frequently be able to cut short the relapse at once. In case there is persistent discomfort in the joints, small doses may be kept up for some time.

[One other salicylic compound deserves mention—the oil of wintergreen; this has been used largely by Kinnicutt, who finds it efficacious, easy to take, and not likely to produce the unpleasant symptoms which sometimes follow the compounds in more general use. It is given in doses of 10 to 15 minims every two hours, either in milk, on sugar, or in capsules. Salicin is well borne even by children, but it is bitter. The dose is 10 to 40 gr. (gm. 0.6 to 2.5).

It will be noticed that the author does not mention alkalies. They have been largely superseded by salicylates. Some practitioners combine the two, giving enough carbonate of sodium or citrate of potassium or of sodium to make the urine alkaline and keep it so. Lee asserts that in this way larger doses of the salicylates can be given without toxic effects, provided constipation be prevented. Probably most cases of acute rheumatism get along as well without alkalies, but in obstinate cases, or if cardiac complications develop, they are advisable. "In acute rheumatic affections of the heart," says Mackenzie, "the use of the salicylates is often attended by great benefit. These should, however, be pushed."]

The efficiency of salicylic preparations in articular rheumatism is, unfortunately, limited. Though the course of many cases is rapid and favorable, we may still observe many in which salicylates either fail altogether, or soon lose their efficacy. Such cases are often very trying to the patience of the physician as well as to that of the sufferer. Salicylic acid is frequently entirely ineffective in cases in which the disease fastens itself in one or a few joints. In such cases, we partially or entirely abandon the use of the salicylates for a time, and adopt a different treatment. Not much is to be expected from internal medication. External treatment is more effective; above all, Bier's hyperæmia (rubber bandages above the affected joints, one fourth to one half hour or longer, daily), also local application of hot poultices, applications of thirty-per-cent alcohol, etc. The external application of penetrating salicylic preparations (mesotan, rheumasan, salit) may also be tried, but care is necessary to prevent inflammation of the skin, which may readily ensue. Where the disease has reached a more chronic stage, general and local sweats (hot-air baths, electric-light baths), packing with hot peat, fango, etc., are to be considered. Galvanization and massage of the joints may also be useful. We should warn against the too early use of warm baths, as they often excite renewed pains in the joints.

General hygienic and dietetic treatment should not be undervalued. An equable temperature should be carefully maintained in the sick-room, inasmuch as cold, or draughts, or moisture have very often been found to exert an evil influence upon the disease and excite fresh pain. The patient should therefore be kept warm, and it is sometimes advantageous to wrap up the affected joints in cotton batting. It is often advisable to apply splints to joints that are more severely affected. It is of great importance that even in the mildest cases the patient should be strictly confined to bed, and he should by no means get up too soon. If possible, we keep our own patients in bed for a week after the pain has ceased. Getting up too early will very often bring on a relapse. With regard to diet, milk is the best food. We may also allow soup, eggs, and a little meat. In France, great weight is laid upon an exclusive milk diet; but this would seem to us an extreme view.

We do not need to speak at length about the treatment of the complications and sequelæ, since we should merely repeat what has already been said in the appropriate chapters of this work. There has been much said on both sides as to the influence of salicylates in preventing complications, particularly cardiac complications. This much is certain, that cardiac complications are not absolutely prevented by the salicylic treatment, and that they too frequently occur while it is being employed; but we do believe that this treatment decidedly shortens the course of the disease as a whole, in many instances, and thus lessens the liability to endocarditis. If, however, a cardiac complication has made its appearance, salicylic acid does not exert any appreciable influence upon it.

Another important question is in regard to the efficiency of salicylic acid in the graver forms of articular rheumatism, particularly in cerebral rheumatism. It may be stated, in the first place, that cerebral rheumatism has apparently become much less frequent since the salicylic treatment was introduced. Before the introduction of the salicylic treatment, at least one case of hyperpyrexia occurred each year in Leipsic. Among the many hundred cases seen by us since that time, we have only extremely rarely observed instances of hyperpyrexia. We had an opportunity to observe a case in which hyperpyretic symptoms had already appeared when we first saw it, and which had not been treated with salicylic acid. Here large doses of that remedy produced no effect. We should, nevertheless, be inclined to employ it, first of all, in such cases; and the energetic use of cool baths would probably be the most speedy way of modifying the dangerously high temperature. Stimulants, in particular camphor, are also required in the severe cases.

In the severe hemorrhagic varieties of rheumatism we should also give salicylates a trial. The milder hemorrhagic cases (hemorrhagic urticaria) require no special methods of treatment.

If the acute affection merge into a chronic condition of stiffness and swelling of certain joints, such as the wrist or shoulder, we must employ the same remedies as in chronic articular rheumatism.

The best results are obtained from Bier's hyperæmia, the local application of heat (hot air, fango, incandescent electric lights), general sweating processes, massage, passive movements, galvanic current, etc. Warm baths may also be ordered in such cases (see the following chapter). The patient might be sent to Teplitz or Wiesbaden. These baths are good resorts for convalescents. The subsequent muscular atrophies and paralyses recover most rapidly under electricity.

Prophylaxis requires, first of all, that one should avoid cold or wet, and other "rheumatogenous influences." Persons who have already had one attack of articular rheumatism must be especially careful, inasmuch as they are more than ever liable to the disease, as has already been said. It is not inconsistent, however, with the exercise of due caution, to endeavor to lessen the sensitiveness of the skin by such procedures as cold sponging, followed by friction. [The tonsils, being a possible gate of entry for the disease into the system, should be examined, and if diseased be treated or enucleated. A history of recurring tonsillitis establishes the need of interference. Adenoids also may favor rheumatic infection.]

CHAPTER II

CHRONIC POLYARTHRITIS—CHRONIC ARTICULAR RHEUMATISM

Definition and Ætiology.—Our knowledge in regard to the nature and causes of chronic polyarthritis is still very meager, despite the great frequency of cases belonging to this class. Disregarding tuberculous and gonorrheal diseases of the joints, with their established ætiology, in almost all other multiple articular affections the underlying cause can at best only be surmised. In a number of affections belonging to this class the assumption appears natural that we have to deal with the same morbid process, only in a chronic form, as in acute articular rheumatism. For such cases the old name of "chronic articular rheumatism," still so frequently used, is entirely appropriate. The ætiological relation between acute and chronic articular rheumatism is shown most distinctly in those cases in which a persistent chronic joint disease is a direct sequel of acute articular rheumatism. It is hardly possible to doubt that the same specific poison which excited the acute arthritis maintains possession of the joints, and produces the chronic inflammatory changes. Cases of this sort are not very infrequent, and may be of slight or great severity. The worst cases produce changes which fully justify the appellation of arthritis deformans, although this term is used merely from the gross appearances. But the cases which develop from what is clearly acute articular rheumatism, are, after all, rare in proportion to the great majority, which from the first develop very slowly and insidiously.

We may well suspect that the same injurious agents are instrumental to some extent as in acute polyarthritis, but we have no positive proof. We may be the more inclined to speak of a "chronic rheumatic polyarthritis," where we can assume that the so-called "rheumatogenous influences" have probably been instrumental in the production of the affection. Such causes are exposure to cold and wet, and working in cold or draughty places, or dwelling in newly built and damp houses. This explains why those who follow certain callings—for instance, washerwomen—are more apt to suffer from the disease than others, and why arthritis deformans has, with some justice, been called a disease of the poor, in contrast with the gout of the wealthy. The term "chronic arthritis deformans" is not an ætiological, but an anatomical one. Its purpose is to point to the marked "deforming" changes in the joints, and, therefore, is applied only to the more severe types of the disease, the milder ones without any considerable anatomical disturbances being designated merely as "chronic polyarthritis," or, in accordance with what has just been said, as "simple chronic articular rheumatism." Various diseases, which ætiologically are entirely dissimilar, are very probably grouped together as "arthritis deformans." It is important, above all, to distinguish between chronic gouty diseases of the joints, which may also lead to the most pronounced deformities of joints, and nongouty ("rheumatic") arthritis. As a rule, this distinction is easy (*vide* chapter on Gout), but many cases are very difficult to classify. Chronic gonorrheal diseases of the joints are, of course, to be eliminated as forming a special group. Much has been written in regard to chronic syphilitic disease of the joints, but, in fact, we know very little concerning it. As we shall see later, tabetic joint affections, which are of such practical importance, belong perhaps to this class. Various authorities have maintained that arthritis deformans is the result of a primary disease of the nervous center, and in particular of the spinal cord. We regard this statement as entirely unwarranted. It originated at a time when there was a tendency to ascribe all sorts of ills to disease of the "trophic centers," but there is no doubt that this tendency was carried very much too far. We may state in this connection that a careful microscopic examination of the spinal cord in one case of very severe arthritis deformans yielded an entirely negative result.

[Some striking cases are reported by Blake, which go to show that the arthritic changes may be set up by chronic, though slight, suppuration, in analogy, perhaps, with the synovitis of gonorrhea. Careful examination should consequently be made of all the mucous membranes, especially in cases which seem to have no connection with true rheumatism.]

Chronic arthritis is mainly a disease of advanced years. Certain monarticular varieties, about whose ætiology; it must be confessed, we know little as yet, have been termed arthritis senilis—in particular, the *malum coxæ senile*. Even the common and, in a certain sense, typical form of arthritis deformans (*vide infra*) is not apt to occur in people under thirty-five years of age. This rule, however, has exceptions. Certain forms of polyarthritis are not rare in younger persons between the ages of twenty and thirty years, and we have ourselves seen a few perfectly characteristic cases of arthritis deformans in children between ten and fifteen years of age. Women are much oftener attacked than men. It is often said that trouble, anxiety, and other emotional influences favor the outbreak of the disease, but the proof of this is lacking. The disease does not often seem to be hereditary.

Pathology.—The process is described is simple chronic arthritis (or synovitis) so long as it is confined mainly to the synovial membrane of the joint and the periarticular connective tissue. These parts often undergo decided inflammatory thickening (*synovitis hyperplastica*), the synovial membrane becomes cloudy, and the amount of synovial fluid is more or less increased (*synovitis serosa*)—that is, we have chronic dropsy of the joint. Sometimes different parts of the synovial membrane are connected by adhesions, which considerably interfere with the movements of the joint. There may even be complete anchylosis; for example, in the shoulder or knee.

Chronic synovitis may pass imperceptibly into arthritis deformans. In this, not only the capsules of the joint, but the articular cartilages and the articular extremities of the bones, are so much affected as to produce the most striking deformity. These changes almost always originate in the articular cartilages. In many cases the morbid process consists merely in a fibrillation, destruction, and wearing away of the articular cartilage, beginning usually on its outer edge; finally, the bone is exposed and itself undergoes ulceration. During all these changes the articular capsule may escape involvement, and there may be no serous effusion into the joint (*arthritis sicca*). In other cases, however, there are soon added proliferative changes affecting not only the cartilage itself, but also the synovial membrane. The inner surface of the synovial membrane becomes tufted, and many of these tufts may undergo secondary calcification, break off, and become loose bodies in the joint. The articular cartilages are also affected by inflammatory new growth and proliferation; these in part become disintegrated, and lead to deeper and deeper excavations of the cartilage. From the cartilage the process extends to the bones, and thus at last the articular extremities of the bones become almost completely destroyed. Sometimes, also, the periosteum near the joints undergoes ossific periostitis. Very often the process extends to the neighboring tendons, tendon sheaths, synovial sacs, etc. On microscopic examination of the diseased joints, we find fibrous disintegration of the matrix of the cartilage, and proliferation and subdivision of the cartilage cells, at the same time that there is destruction of the newly formed cells by simple or fatty degeneration.

Of course the normal structure of the joint is at last completely destroyed by these various processes. The articular extremities of the bones waste away more and more, and take new relative positions, as the parts which impinge upon each other are worn away. Externally, the joint usually becomes larger and larger, and this is the more evident because the surrounding muscles undergo great atrophy. There is often no collection of synovial fluid in the joints during the entire illness, but sometimes the joints are swollen—e. g., the knee joint—and even to a considerable degree. There is a tendency for the affected joints to suffer more or less complete anchylosis and contracture (*vide infra*).

Symptomatology.—The clinical symptoms of chronic arthritis are very simple and uniform. They relate almost exclusively to the local disturbances, and to these they are directly due. The general course of the disease, however, presents great diversities. Often the arthritis limits itself to a single joint or to a few joints; in other cases almost all the joints become finally involved.

Except in the cases which are preceded by acute articular rheumatism, the disease usually begins quite gradually and insidiously. The first subjective symptoms are stiffness and pain in the joints, the pain being aggravated by pressure or movement. The stiffness is most noticeable when the joint has remained quiet for some time previous, and is therefore ordinarily greatest on waking up in the morning. The pain often shoots from the joints upward and downward, and is of a burning character, or less often neuralgic. Even in advanced cases the pain usually occurs only when the affected joints are moved, although then it may be very severe. When the body is entirely at rest there is little or no pain. Besides actual pain there are apt to be at the onset of the disease peculiar paræsthesiæ and other nervous symptoms, particularly in the fingers. Motion is impaired very early. This is due at first to the pain, and to a certain reflex inhibition and ataxia of the muscles, to which are later added the purely mechanical hindrances and the ever-increasing atrophy of the muscles.

The objective changes in the affected joints begin to appear soon after the symptoms just mentioned, at least in cases of any severity. The joints often seem enlarged and thickened. If we attempt to move them, we not only cause pain and meet with mechanical obstruction, but we may hear and feel the cracking and grating produced by the rubbing of the denuded and uneven surfaces upon each other. This is often noticed by the patient himself. In chronic arthritis sicca there may be no great swelling of the joints; still, there are usually marked pain, crepitation, and impairment of motion. In other cases, however, there are considerable serous effusions into the joints, usually most marked in the knee joint. The resulting swelling of the joints often varies considerably. At times the exudation is absorbed, only to reappear, and frequently in increased amount.

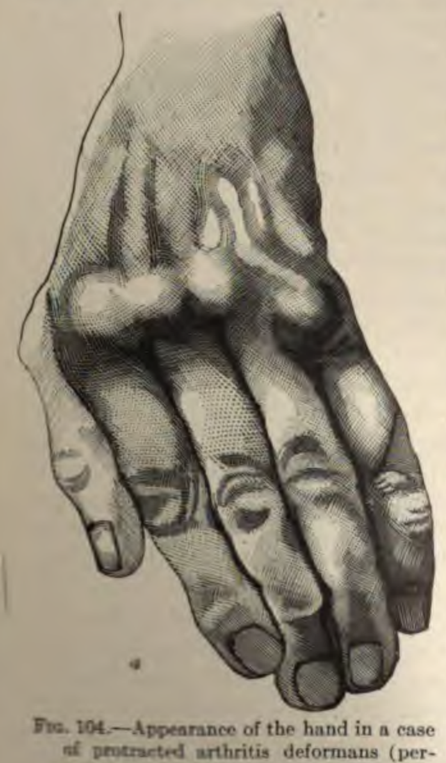
FIG. 104.—Appearance of the hand in a case of protracted arthritis deformans (personal observation).

As the disease gradually progresses, there are usually developed certain characteristic deformities, which are apt to be most strikingly exhibited in the hands (see Fig. 104). The metacarpo-phalangeal joints are thickened and swollen, and are made all the more prominent because the interossei upon the back of the hand are atrophied. The bases of the first phalanges are directed obliquely toward one side, so that the fingers assume more and more the appearance of subluxation. They are bent over toward the back of the hand, and are also displaced toward the ulnar side, so that they often actually come to rest one upon the other. The palm of the hand is frequently deeply hollowed out. Often the phalangeal joints also are distorted, so that, for example, there will be an obtuse angle on the dorsal surface of the fingers between the first and second phalanx, while the terminal phalanx is apt to be flexed, although the second phalanx preserves a position of extension. Despite these changes, many patients, if only the thumb remains tolerably movable, are able to use their hands for quite delicate work, although at the expense of much time and effort. Arthritis deformans scarcely ever causes anchylosis of the small joints of the fingers. The elbow joints are often decidedly enlarged. The forearms usually assume a position of pronation, combined with more or less flexion. Even the mobility of the shoulder joints progressively diminishes, so that at last the arms cannot be raised at all. At the same time there is contracture of the adductor muscles.

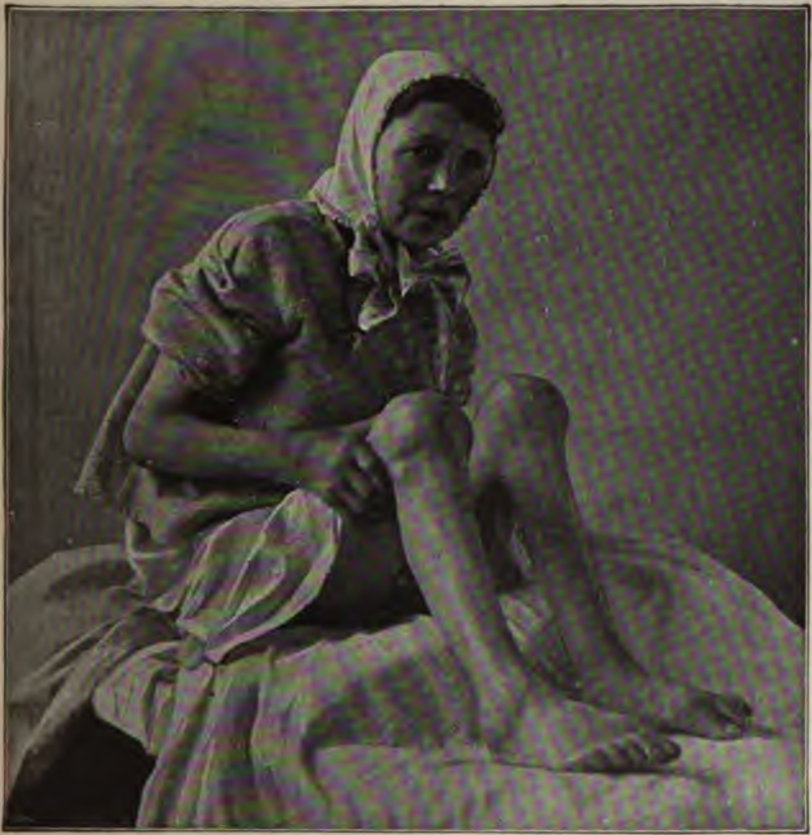

FIG. 105.—Chronic arthritis deformans with contracture of the hip joint and knee joints.

With regard to the lower extremities, the hip and knee joints usually suffer more than the ankle. The small joints of the toe, as a rule, escape entirely. In the larger joints there is sometimes a chronic anchylotic form of arthritis with atrophy of the ligaments present, and in other cases there is more of a hyperplastic condition with exudations. Sometimes there are thickenings of the joints and serous effusions, and sometimes arthritis sicca. If the joints of the lower extremities are much involved, of course standing and walking grow more and more painful and difficult, and finally require crutches, or an assistant. Very often there is marked contracture of the flexor muscles of the hip, and still oftener of the knee. This is not associated with anchylosis of the joints, but is of purely muscular origin. In such cases the patient cannot sit in bed with the knees extended (see Fig. 105). There is sometimes subluxation of the hip joints, so that the head of the femur slides backward and upward. The same thing is observed also in very severe cases in the knee joint.

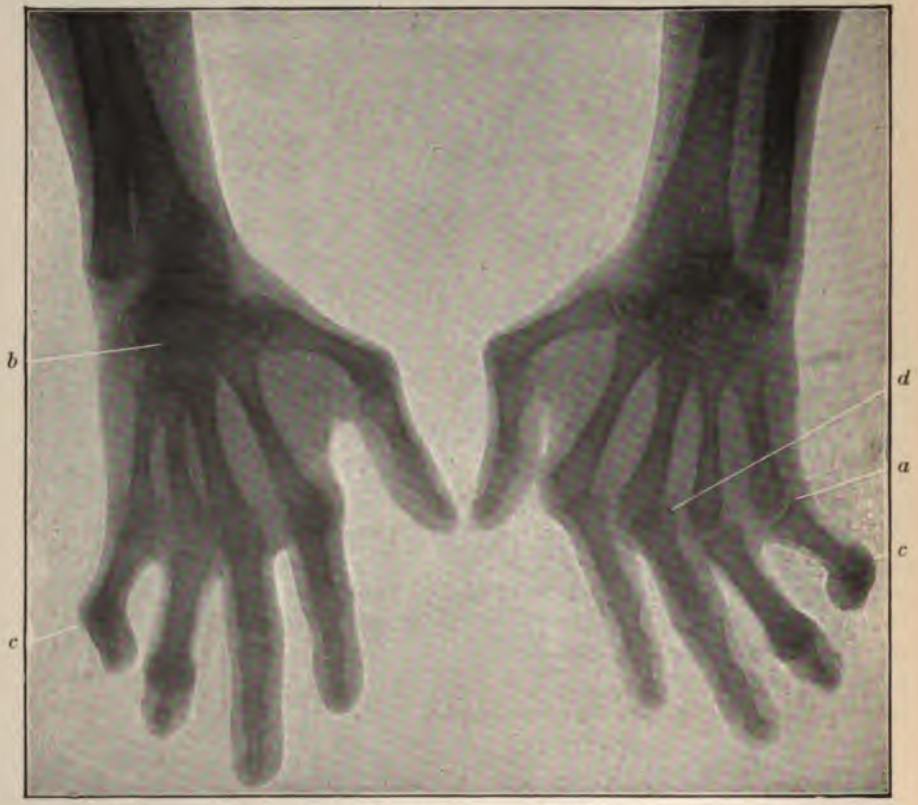

FIG. 106.—Röntgen photograph of the hands in a case of arthritis deformans. *a.* Widening of the joint spaces. *b.* Indistinct outlines of the several wrist bones in consequence of thickening and deformity. *c.* Distortion of the phalanges. *d.* Subluxations.

Examination of the affected joint by means of the Röntgen ray furnishes very valuable information as to the character and extent of the disease (*vide* Fig. 106). It shows very clearly that at first the process affects only the ligaments and capsules of the joint. It is only in the later stages that the joint cartilage becomes thickened, tufted, and fissured, and then structural changes in the articular extremities (osteoporosis, etc.) invariably manifest themselves. There are monarticular and polyarticular forms of the disease. The monarticular form is usually regarded as a surgical trouble, and is most often located in the hip joint (*malum coxæ senile*), or, more rarely, in the knee and shoulder joints.

The polyarticular form is the characteristic one. In most of the typical cases it begins in the small joints of the hand and fingers. At a later period the larger joints are also invaded, one after the other, the invasion taking place symmetrically on both sides of the body, although the disturbance is not infrequently greater on one side than on the other. In severe cases the joints of the spinal column also are involved (compare the Appendix to this chapter). This impairs particularly the movement of the head. The articulation of the lower jaw is usually very little affected, if at all.

There is hardly any affection of parts of the body other than the joints in arthritis deformans. The muscles should be excepted, for they always undergo that arthrogenetic muscular atrophy which we have already described (*vide* page 7), and which is always demonstrable in arthritis deformans, and generally appears strikingly early. This atrophy is most marked in the interossei, the shoulder muscles, and the muscles of the calf and thigh. The associated muscular contractures are also of great importance. These are certainly to some extent connected with chronic inflammatory processes in the tendons and the fasciæ. Sometimes the skin over the wrist and other affected joints appears peculiarly lax and flabby, but frequently, at the same time, smooth and atrophic. The internal organs almost always perform their functions in a perfectly normal manner. Appetite and digestion remain good, although there is often some tendency to constipation. Rarely there is valvular disease of the heart, but usually only in such cases as originate in an acute articular rheumatism. Still, we have seen valvular disease, exceptionally, in cases which had been chronic from the start, a fact which is not without interest from an ætiological point of view, indicating a relationship to the acute polyarthritic process. Certain attendant symptoms which are sometimes observed, such as headache, congestive states, psychical depression, and bronchitis, probably are not directly connected with the disease, but are easily explicable sequelæ.

General Course of the Disease.—Arthritis deformans is an extremely chronic trouble. It may last even ten or twenty years, or more. Sometimes there is an apparent arrest of the process extending over months, or even longer. Sometimes the progress of the disease is marked by remissions and exacerbations, affecting either the general or the local manifestations. In general, however, the disease continually advances.

Prognosis.—The prognosis of chronic polyarthritis and arthritis deformans is therefore unfavorable. Recovery, if it ever occurs, is extremely rare, and is possible only in the early stages. For the encouragement of the patient, it may be said that, under proper care and treatment, the disease often runs so gradual a course that the general condition remains at least bearable for a very long while, although there may be considerable local disturbance. The disease is not directly dangerous to life. The eventual fatal termination ensues either from general debility or because of some intercurrent disease.

The prognosis is somewhat more favorable in the milder cases of "chronic articular rheumatism," where the anatomical changes are less severe, and are completely limited to the synovial membrane. Even here, however, recovery is by no means frequent, and it is always to be feared that grave deformities of the joints will gradually be developed.

Treatment.—With regard to regimen, it is requisite in the first place to avoid all unfavorable external influences. If possible, the dwelling should be dry and warm, and it may often seem advisable to make a change of climate. The patient must dress warmly, without, however, undermining his powers of resistance too much, as he will be in danger of doing. The diet must be abundant and nutritious.

Internal remedies may be tried with the hope of modifying the disease, but our chief reliance must be upon local treatment of the joints. Among internal remedies the most important are the salicylates. The persistent use of small doses of salicylic acid or salicylate of soda, 30 to 45 gr. (gm. 2 to 3) a day, and similar remedies, is recommended by many physicians. We have ourselves seen no special advantage from their employment, and therefore usually prescribe them only during acute exacerbations of the disease. Among other drugs that are recommended are iodin and arsenic. Iodin may be given in the tincture (a few drops in mucilage several times a day), or, better, in the form of potassium iodid, or sajodin. As yet we, ourselves, have not seen any great benefit from iodin, but we have in repeated instances witnessed an apparent improvement from the use of arsenic. It is best administered in pills containing $\frac{1}{30}$ to $\frac{1}{15}$ gr. (gm. 0.002 to 0.004) of arsenious acid, one pill two or three times a day. If this remedy prove beneficial, it must be continued for at least months, perhaps with occasional brief intermissions. Iron, quinin, and cod-liver oil are sometimes indicated by the general condition. First among local methods of treatment are hot-air treatment and Bier's hyperæmia. General as well as local sweating processes, by means of hot-air chambers, incandescent electric-light baths, etc., undoubtedly produce quite good results in some cases. Then follow local moist-hot applications and packs (peat, fango, various kinds of mud, etc.). Bier's hyperæmia, one to two hours daily and longer in various gradations, may also have a favorable effect, but of course too great hopes must not be placed upon it in severe cases. In addition to these we have massage, although the good it accomplishes is, of course, apt to be evanescent. It will, however, do much to hasten the absorption of inflammatory exudations, and also to loosen up the joints, invigorate the muscles, and improve the general health. The Swedish-movement cure will be found of great benefit in all cases if begun early and methodically persevered in. It preserves the mobility of the joints as long as anything can. Electricity also has a beneficial influence. The galvanic current is applied to the affected joints, and the faradic current to the atrophied muscles.

Baths are universally employed in chronic arthritis. Their value should not be overestimated, but, notwithstanding, their usefulness is undeniable in many cases. Simple warm baths, or salt baths (5 to 10 pounds of salt for each bath), are practicable in almost any household. As health resorts in arthritis deformans, experience shows the following to be most desirable: The simple warm baths, such as Teplitz, Wildbad, Ragatz, and Baden in Switzerland; the warm chlorid-of-sodium baths in Wiesbaden; the acidulated baths of Oeynhausen and Nauheim—all have been found most useful in arthritis deformans. Quite good results are also obtained, particularly in the earliest stages, from the moor baths or peat baths of Elster, Marienbad, Franzensbad, Aibling, etc., as well as from the various mud baths (fango baths, etc.). Sulphur baths—Baden near Vienna, Pöstyèn (Pistyan) in Hungary, and Trencsin-Teplitz in upper Hungary—are also recommended by many.

[The mineral springs within the limits of our own country chiefly to be recommended are Sharon and Richfield, in New York State; the Sulphur Springs and Hot Springs of Virginia, and the Hot Springs of Arkansas. There are also Santa Rosalia in Mexico, and Banff in Canada.]

We have repeatedly seen quite excellent results follow the employment of hot sand baths. These also can be easily used at home, particularly if applied merely to the hands or feet. They are employed more elaborately in Köstritz and Blasewitz. These hot sand baths seem to do good, not only from the temperature, but also from the uniform and persistent compression which they exert.

Stimulating or narcotic remedies may be rubbed into the joints, but they are beneficial only because of the massage which accompanies their employment. In practice it is not always possible to omit their use. We have never seen any special benefit from ichthyol. The application of tincture of iodin is usually entirely without effect. As to morphin and other narcotics, the disease is so chronic that it is desirable to employ them as little as possible. A considerable number of those who suffer from chronic arthritis become opium eaters. We should try, therefore, if there is severe pain, to give relief as long as possible with antipyrin, aspirin, and similar remedies. Local applications of penetrating salicylic preparations, such as mesotan, rheumasan, and salit, may be cautiously tried.

We may say, therefore, that the use of the various remedies which have been suggested one after another will enable us to oppose some obstacles to the progress of the disease. Persistent treatment will, in many cases, be rewarded by considerable, at least temporary, improvement.

APPENDIX

CHRONIC ANCHYLOSING RIGIDITY OF THE VERTEBRAL COLUMN

(Strümpell-Marie's Disease) [*Spondylitis deformans, Strümpell-Marie Type*]

In connection with the chronic arthridites, we will now discuss a peculiar disease of the vertebral column which has not infrequently been observed in middle-aged men. The course of this disease is very tedious, and generally quite painless, and it may ultimately lead to a complete rigidity of the vertebral column, which then becomes an absolutely rigid rod from the os sacrum to the occipito-atloid articulation. All motion of the trunk and of the head, as well as sitting and lying, are impeded to a considerable degree; standing and walking not to so great an extent. In the less advanced cases the rigidity is not so complete. The latter usually begins in the lumbar portion of the vertebral column and thence advances upward; a downward extension is much less frequent. In most cases the general form of the vertebral column is rather straight. In fact, even its normal curvatures, and especially the normal lumbar lordosis, become somewhat reduced. There are, however, cases with pronounced kyphotic curvature (Bechterew). These differences probably have some relation to the previous condition of the vertebral column, as well as to the occupation and work of the patient. In consequence of their inactivity, the muscles of the back, and particularly the long spinal muscles, atrophy. We personally have never observed a pronounced participation of the nerve roots. Occasionally, however, we may observe neuralgic pains, muscular contractions, and the like, which may perhaps be due to an irritation of the spinal-cord roots.

It is very remarkable that very often the process is limited to the vertebral column, and leaves all the other joints free. On the other hand, the joints of the extremities are also not infrequently affected at the same time. Similar anchylotic or apparently chronic arthritis changes appear, above all, in the large proximal joints (hence the term *spondylose rhizomélique*), but also, though less frequently, in the other joints. In the first case, which I observed more than thirty years ago, besides the complete rigidity of the vertebral column, there was a total anchylosis of both hip joints, so that the gait of the patient was peculiarly affected; he walked, as it were, on his knees.

The anatomical process in vertebral anchylosis is not always exactly the same. We have to do substantially with a bony anchylosis of the articular processes of the vertebræ; in most instances there is also an ossification of the ligaments, so that the individual vertebræ are joined together, anteriorly or laterally, by thick osseous bands. Such bands may also be formed between the spinous processes. The articulations between the vertebræ and the ribs are also frequently anchylosed, and this may lead to a change in the type of the breathing. The intervertebral disks may escape for a long time, but finally they also may become ossified.

The true nature of the disease has not been fully cleared up. We commonly speak of a "chronic anchylosing spondylitis," but possibly we should distinguish between the "chronic-inflammatory" and the simple "metaplastic" processes.

As in ætiology, we know a single factor of undoubted importance, viz., trauma. With noticeable frequency we obtain a history of a fall upon the back or of being crushed from above, or similar accident. Often it is some years after the trauma before any marked changes can be demonstrated.

The diagnosis is not difficult, provided due care is exercised and a thorough examination made. The Röntgen ray is very helpful, showing clearly the bony union between the separate vertebræ, as well as any alterations which there may be in the intervertebral disks.

Cure of the condition is impossible, but some symptomatic improvement may be obtained by means of baths, the cautious use of medical gymnastics, massage, and the like.

CHAPTER III

ACUTE AND CHRONIC MUSCULAR RHEUMATISM

(*Myositis or Myalgia rheumatica*)

Definition and Ætiology.—Certain acute affections may originate primarily in the muscles. These are to all appearance inflammatory in their nature, and not infrequently result from taking cold, or other causes similar to those which produce acute articular rheumatism. These affections are classed as "acute muscular rheumatism" or acute rheumatic myositis. It is possible that these diseases also are infectious, but the question remains entirely undecided. The analogy which both these troubles bear to acute articular rheumatism is not complete. It is seldom that the two processes are seen in combination, and, furthermore, acute myositis is not "polymuscular," but is usually confined to one muscle or to a single group of muscles; it is attended with little, if any, fever, and it is almost never followed by acute endocarditis. The two diseases, therefore, are alike only in certain symptoms (pain and impairment of motion), and in the fact that they are often, although not always, ascribable to cold or the like.

Besides genuine acute myositis, there are many other cases in which pain suddenly occurs in the muscles ("myalgia") without any attendant objective change. It is sometimes difficult to know how to regard these cases. In practice they are usually termed muscular rheumatism, especially when they are referable to rheumatogenous influences, and it is possible that many such cases are really a very mild form of the genuine inflammatory disease. Yet in our opinion it is perfectly possible that some chemical or physical changes in the substance of the muscle (perhaps an alteration in the general state of the muscle-plasma, abnormal conditions of coagulation (?)) occasion the local disturbance. That the influence of cold is often, though not always, important in this connection seems to us indubitable. There are individuals who are almost certain to have a stiff neck if they are exposed to any decided draught upon the back of the neck, and others who, every time they get chilled, are liable to lumbago. However, other injurious influences may, of course, be important. Thus, traumatic pain in the muscles is the result of some excessive strain, and in many instances is apparently due to laceration of some of the muscular fibers. This is generally occasioned by too violent muscular exertion. Any physician who sees many patients from the laboring classes meets with an abundance of cases of this sort.[1]

The limitations of acute muscular rheumatism are obscure, but still more so are those of "chronic muscular rheumatism." This disease also is a frequent one, and only imperfectly understood. It does not bear a close analogy to chronic articular rheumatism except in this unimportant point, that chronic muscular rheumatism seems to be quite often occasioned by meteorological influences. While the anatomical changes in chronic articular rheumatism are almost always striking, analogous lesions are very exceptional in chronic muscular rheumatism. On the contrary, the name is usually applied to cases where there is pain in various muscles all over the body, but where there is no discoverable objective disturbance. Older authorities used to speak of "rheumatic induration" of the muscles, but this or any other actual anatomical change is very exceptional.

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[1] Some time ago the author saw an organist who had to work the pedals for many hours a day; he had an extremely painful affection of the lower extremities associated with swelling, which could be regarded only as an acute myositis.

These facts justify a doubt as to whether all cases of chronic muscular rheumatism actually deserve their name. It is certainly quite appropriate in those not infrequent cases which seem to be due to "rheumatogenous influences," and are so evidently aggravated upon every exposure to cold, or every period of bad weather, that the patient often asserts that he carries in his bones the best of barometers. Such is the "old rheumatism" of those who have passed a large part of their lives in the open air, regardless of wind or weather. There are other cases, the character of which is different. In them the muscular pain is associated with general constitutional conditions such as gout, anæmia, etc., or with corpulence (when it is, perhaps, the result of circulatory disturbance), or possibly with chronic poisoning. An important instance is the "rheumatic pain" sometimes complained of by topers, which we are inclined to ascribe not to changes in the muscles, but to nutritive disturbances of the nerves. Finally, one must bear in mind those many indefinable pains met with in hysterical and nervous patients, for which the doctor can find no objective cause. For these and similar disorders there are no special names, and the practicing physician often terms them all "muscular rheumatism," a diagnosis with which the patient is usually quite contented.

Clinical History.—Genuine acute muscular rheumatism is ordinarily, as has been said, limited to some one definite group of muscles. The affected muscles often seem somewhat swollen and infiltrated, are very sensitive to pressure, and, if not quite useless, are nearly so, greatly impairing the motion of the corresponding member of the body. All these symptoms are best illustrated in acute myositis of the deltoid (omalgia). The whole shoulder is swollen, the muscle is very painful, and the upper arm is almost incapable of voluntary motion, although, if caution be exercised, passive movement can be made without causing any pain.

The various forms of acute muscular rheumatism have received names descriptive of the locality of the affection. We have, besides the omalgia already mentioned:

1. *Rheumatism of the cervical muscles* (splenius, trapezius, sterno-cleido-mastoid, etc.), *Myalgia cervicalis, or rheumatic torticollis.* The pain is located in the back and side of the neck, and is usually unilateral. The head is held stiffly to one side, the occiput inclines toward the painful side, and the chin is turned toward the healthy side. All active and passive motions which tend to stretch the painful parts are much impaired and cause suffering.

2. *Myalgia lumbalis, or lumbago.* This, the most acute form of muscular rheumatism, known among the common people in Germany because of its sudden onset as "witch's shot," or "dragon shot," is a well-known disease. The region of the sacrum and loins in a greater or less extent (erector spinæ, quadratus lumborum, etc.) is very sensitive. All motions of the trunk, such as bending and twisting, are very painful and difficult. The disease is more frequent in men than in women. Certain persons seem to be especially pre-

disposed to it. It should also be stated that lumbago is not always of a rheumatic character, but of traumatic origin, as from lifting a heavy weight, or from sudden stooping.[1] [It is very common in the traumatic neuroses, where it was once regarded as indicative of spinal injury.] On the other hand, a latent disease of the muscle may often be made active through such a cause.

3. *Rheumatism of the thoracic muscles,* and particularly of the intercostals. This may cause great discomfort, as it renders breathing, coughing, and sneezing very painful. It is comparatively rare, and caution should be exercised in diagnosticating it, to avoid confusion with pleurisy and periostitis of the ribs. Very often, also, thoracic disturbance is regarded as rheumatic when it is really traumatic, being the result of stretching or laceration of the fibers of the pectoral or other muscles.

4. *Rheumatism of the head* also, probably, belongs in this category, although the affection is seldom confined to the muscles of the scalp, but involves also the fasciæ, and may even be almost confined to them. It is not infrequently excited by exposure to cold. The pain is quite violent, and greatly increased by any movement of the scalp. Of course, the diagnosis requires the previous exclusion of the various forms of headache described elsewhere in this work.

The duration of acute muscular rheumatism is brief. Usually the pain abates in a few days, but a tendency to relapse persists for some time. Constitutional symptoms in addition to the local disorder are, as a rule, absent or slight; still, we sometimes meet cases in which acute muscular rheumatism is associated with fever and marked general disturbance.

In chronic muscular rheumatism there are, as a rule, no objective lesions discoverable. The statements of masseurs as to palpable nodes and indurations are, in our opinion, to be received with very great skepticism. The pain is seldom located permanently in any one place, but it is felt first here and then there. It is usually increased during bad weather, and is less severe when the weather is warm. The pains are often described as "wandering." Motion is seldom much impaired. Sometimes, however, there may be a certain stiffness of the muscles, most marked after a period of repose.

The diagnosis of chronic muscular rheumatism rests, therefore, mainly upon the rational signs. Hence, it is often impossible to avoid the suspicion of malingering, particularly where certain applicants for hospital care are concerned. We should not, however, be too uncharitable, since without doubt there are cases in which quite severe pain is felt, now in one set of muscles and now in another, without any anatomical basis for such pain being discoverable. Nor should we ever forget that other diseases may have pain for their first symptom. It is not at all exceptional for the lancinating pains of locomotor ataxia to be for a long time regarded as "rheumatic." Lumbago may be confounded with insidiously developing tumors, diseases of the vertebræ [irritation of the sacro-iliac synchondrosis], renal calculi, or with various abdominal disorders (particularly in women). We should therefore never omit to make a careful physical examination.

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[1] Lately, S. Erben has sought to prove that lumbago and torticollis are not at all muscular diseases, but probably in most cases due to an affection of the joints of the vertebræ. He bases this belief upon the result of painstaking investigations. One who, like the author of this textbook, has often himself suffered from acute muscular rheumatism, will be scarcely inclined to adopt this view, for the subjective sensation of pain is referred decidedly to the muscles, or to the attachments and fasciæ of the muscles.

Treatment.—Acute muscular rheumatism has this in common with acute articular rheumatism, that it is usually very favorably affected by the salicylates. In cases of genuine acute rheumatic myositis, their employment in the manner already described will often give surprising relief in as short a time as twelve or twenty-four hours. We also see good results from antipyrin or salipyrin, etc. Local treatment of the affected muscles may also be followed by great and speedy improvement. Massage is particularly valuable. It is not infrequently the case that a single properly conducted massage may cause a violent lumbago or omalgia to improve rapidly, and like favorable results are witnessed when there is traumatic pain in the muscles. Most of the external applications which are so frequently prescribed for rheumatism—such as spirits of camphor or chloroform liniment—accomplish less through the cutaneous irritation they produce than by the massage incident to their employment. Next in value comes electricity. Both the constant and the faradic current may be employed. Often, also, the local application of warmth is very beneficial; thus, hot dry cloths or hot poultices may be applied. Simple stimulation of the skin by mustard is often palliative, but less so than the first-mentioned remedies. Often great relief is given by sweating, by means of a hot wet pack, or by a steam bath, the employment of which is so common that patients often use it of their own accord.

In chonic muscular rheumatism the benefit of the salicylates and antipyrin is merely temporary, and therefore is to be sought, if at all, only when there is an acute exacerbation. Massage and electricity are more effective, and, if persevered in for some time, will often accomplish good results even in obstinate cases. Treatment by sweating and bathing is frequently prescribed with advantage. Steam baths are often beneficial, but their use requires great caution when the patient is corpulent and has a tendency to congestion or cardiac failure. There is also value in mud baths, pine-needle baths, saline baths, and in the baths given at Teplitz, Baden-Baden, Wiesbaden, Warmbrunn, and other places.

In many cases of chronic muscular rheumatism constitutional treatment is of great importance. Particularly when the patient is overfed, and intemperate in the use of alcohol, much benefit will often be accomplished by a proper regulation of the ingesta and the prescription of a sufficient amount of muscular exercise. Such patients may also be helped by a cautiously conducted cold-water treatment. This lessens their great sensitiveness to cold.

[In acute cases with localized pain I have found useful a thick flaxseed poultice, applied as hot as it can be borne, renewed once or twice, and followed by the application of a thick layer of cotton. A dry cup or two is also often productive of great relief. In chronic cases, plasters and the iodid of potassium are often of benefit.

Muscular rheumatism is a common and often very troublesome affection in those whose occupation calls for decided muscular exertion. A muscle is strained, pain settles in and is apt to recur in the part; and, while the general health is sufficiently good, the man is compelled to remain idle. Quack advertisements dwell so much upon pain in the back as a symptom of Bright's disease, that we are frequently consulted by those who, suffering from muscular pain and soreness, think themselves the subjects of serious disease of the kidneys.]

CHAPTER IV

ACUTE POLYMYOSITIS

THROUGH the observations of E. Wagner, Unverricht, and others, we have obtained knowledge of a disease which consists essentially in an acute inflammation of most, or even of all, the muscles of the body. The disease appears chiefly in persons in early or middle life, but it has also been repeatedly observed in the elderly. The disease may begin either quite suddenly or gradually. Sometimes polymyositis is a sequel of some other disease, most often, as it seems, of septic infections, such as puerperal fever and erysipelas. This circumstance seems to indicate that polymyositis is associated with severe constitutional intoxication.

The disease usually begins with decided constitutional disturbance, including languor, headache, anorexia, and vomiting. Very soon the characteristic muscular pains commence, there are dragging pains, and often cramps in the arms, legs, and trunk, and these sooner or later decidedly impede motion. If we examine the painful places we find a marked tenderness upon pressure and upon passive motion. Soon we can perceive a distinct swelling of the parts involved. This depends somewhat upon the inflammatory swelling of the muscle, and still more upon a firm inflammatory œdema of the corresponding skin, which usually appears very early. As a rule, the upper extremities are most severely attacked, and in them, too, the inflammatory œdema is most marked. The forearm swells, particularly in the region of the belly of the muscles, and so comes to have a characteristic spindle shape, provided the region of the wrist joint is not involved. The face, the trunk, and the lower extremities also present similar though less marked swellings of the skin, or of the different soft parts, particularly on the extensor surfaces. At the same time, the skin is apt to be distinctly reddened with inflammation, so that it almost suggests erysipelas ("dermatomyositis"). Other exanthems also may appear—viz., erythema, urticaria, and herpes. If the disease lasts some time the swelling gradually disappears, and there may be distinct atrophy of the muscles. The patellar reflex is usually abolished from the start. The electric excitability of the muscles rapidly diminishes, and is finally destroyed. In some few muscles we sometimes observe the reaction of degeneration, but a careful electrical examination is always very difficult because of the great pain it causes. The sensibility of the skin in cases of polymyositis (*vide infra*) remains unimpaired.

The general condition becomes very much aggravated if the muscles of deglutition and respiration are attacked; the ingestion of food is rendered more and more difficult, and there is severe dyspnœa. Soon bronchitis and lobular pneumonia develop, and these are the more distressing to the patient because expectoration grows increasingly difficult and finally quite impossible. Nutrition is often interfered with by stomatitis. Tonsillitis has been repeatedly observed. Perspiration is decidedly increased, and the urine may contain a small amount of albumen. Splenic tumor has been repeatedly found in the acute cases. The bodily temperature is often elevated, 101° to 103° F. (38.5° to 39.5° C.); the pulse rate may rise to 100 or 120 beats per minute.

With regard to the clinical course of the disease, we should make a distinction between the acute and the more chronic cases. The acute may end in recovery after two or three weeks. Often, however, after a shorter or longer course of from three weeks to three months, a fatal termination occurs, probably in most instances the result of respiratory disturbances, secondary pneumonia, and the like. Chronic cases may last a year or more.

In the cases observed thus far there has been found on examination a true acute inflammation of the muscles. Not only do the muscular fibers show all forms of degeneration and destruction, but also in the interstitial connective tissue we find true inflammatory foci (accumulation of nuclei about the vessels, etc.). In a case which we examined these changes were apparently to be found in all the muscles, even in the tongue, the ocular muscles, etc. In genuine polymyositis the peripheral nerves are perfectly normal. Yet recently there have been repeated observations of cases in which a genuine polyneuritis was demonstrated along with genuine polymyositic lesions (*neuromyositis*). These cases indicate a probably close ætiological relation between polymyositis and polyneuritis (*q. v.*). Clinically, the appearance of polyneuritis as a complication may be recognized by slight disturbances in sensation, and by tenderness along the nerve trunks.

We have already noted above that the inflammatory changes frequently extend to the other soft parts and the skin (*dermatomyositis*).

Diagnosis.—The diagnosis of polymyositis is not difficult, on the whole. The pain in the muscles, their swelling, the impairment of motion, the inflammatory changes in the skin, etc., present a characteristic clinical picture. It is important to exclude a possible trichinosis. In this regard we must consider the ætiology, the initial gastrointestinal symptoms, and the œdema of the face. [It is not yet certain whether eosinophilia is peculiar to the myositis occasioned by trichinæ or common to all forms of it.] That there does not seem to be any sharp dividing line between polymyositis and polyneuritis has been already noted. We would like to point out, also, that a group of symptoms very similar to polymyositis is sometimes excited by a peculiar disease first described by Kussmaul and Maier, which is as yet very imperfectly understood. It attacks the small arteries and is termed "periarteritis nodosa." If the smaller arteries of the muscles are attacked with resultant inflammation of the vascular wall, thrombosis, and similar changes, then degeneration of the muscular fibers ensues, with consequent swelling, pain, and impairment of function. At the same time there are usually severe constitutional symptoms, such as fever, anæmia, nephritis, and severe abdominal pain.

Treatment.—In early cases of acute polymyositis trial should be made of salicylic acid and its compounds, antipyrin, and similar remedies; beyond this, treatment must be purely symptomatic, including inunctions of chloroform oil, the administration of narcotics, and the like. Some cases may be benefited by galvanism.

CHAPTER V

RACHITIS

(*Rickets*)

Ætiology.—The first accurate description and the now universal name of "rachitis" (from ῥάχις, the spinal column) is to be ascribed to the Englishman, Glisson, who published a comprehensive monograph upon this disease in 1650. It was his opinion that it first appeared in England, in the beginning of the seventeenth century; and for this reason rickets is still often called by Germans "the English disease." At present rickets is a very widely distributed disease in the North Temperate Zone (especially Northern Germany, Upper Italy, etc.). In the tropics, in mountainous regions, and in the far north it does not occur.

Although the clinical and anatomical phenomena of rickets have been often and accurately investigated since Glisson's time, its true cause still remains entirely unknown. It is certain only that its development is promoted by all unfavorable external circumstances affecting the nourishment and health of the child. It is therefore more frequent among the poor than the wealthy, in damp, cold lowlands than in a dry climate, in the damp, musty, and crowded quarters of large cities than in the country, and among artificially fed, and therefore weakly and anæmic children, than among such as receive the mother's milk. Nevertheless, the essential cause of the disease is not to be sought among these various influences, for rickets undoubtedly does occur, although rarely, in children whose circumstances seem in every respect most favorable.

Guérin, Friedleben, E. Voit, Wagner, Baginsky, and many others have made very exhaustive experimental researches with regard to the development of rickets. It has been found possible to produce certain changes in the bones of growing animals by giving them as little lime as possible in their ingesta, or by administering very large amounts of lactic acid, with the purpose of dissolving the calcium salts, or by giving small quantities of phosphorus. The changes thus caused have been, with more or less correctness, regarded as analogous to those of rickets. These investigations are of great interest with regard to the physiology of bony structures in general, but in our opinion they throw little light upon the clinical question which here concerns us. It is indeed natural enough to suppose that rickets in childhood may be due to an insufficient proportion of lime in the food; or to a defective absorption of the lime salts, on account of intestinal catarrh; or to an abnormally abundant production of lactic acid, or even of carbonic acid, which may disolve the lime salts in the system; but every one of these theories is contradicted by the facts of experience. We know, for certain, that the supply and the absorption of lime salts are absolutely undisturbed in rachitic children; only the utilization of the lime salts for the normal upbuilding of bony tissue is interfered with.

In our opinion, everything seems to indicate that some special, specific, ætiological factor is requisite for the development of rickets. This factor, however, is as yet entirely unknown to us. The assumption of a specific infectious cause is very improbable. We could better suppose that there has been a deviation from the normal bony growth from disturbances in its constitutional regulation by the products of internal secretion. It has often been asserted that heredity plays an important part in rickets. At all events, it is noteworthy that quite often several children of the same family are attacked by the disease.

Rickets is most common in children two or three years old. According to Kassowitz, the disease usually begins in the first months of life, while the severe symptoms do not appear till the second year. Those congenital changes in the bony structure which are designated as "fœtal rickets," have probably no connection with true rickets. Cases of so-called "*rachitis tarda,*" where the disease is said to attack children of from eight to ten years and over, are at all events very rare, if, indeed, their occurrence cannot be altogether denied.

Sex exercises no great influence upon the occurrence of the disease.

Pathology.—Rickets consists of a peculiar disturbance of the processes connected with the growth of the bones. As a result of an increased absorption of the already formed bony tissue, and especially as a result of an insufficient or an almost wholly deficient deposition of lime salts, the bones become or remain abnormally flexible and soft, so that they can easily be cut with a knife.

Upon minute examination, we find both the periosteum and the marrow much reddened and congested. If we try to detach the thickened periosteum from the bone, not infrequently a few bits of bone adhere to the membrane. The most striking changes, however, are exposed upon making a longitudinal section of the bone. They are located at the bases of the epiphyses, because here is the place where the normal, and therefore the abnormal, processes of ossification are most active. Under normal circumstances, the epiphyseal cartilage of the bones in childhood is separated from the main shaft by two narrow layers: first, an outer one, nearest the epiphyseal cartilage, of a bluish color, and 1 or 2 mm. thick; this is the proliferative layer, or hyperplastic zone, where the cartilage cells become divided and arrange themselves in rows. Secondly, an inner, dull yellow layer, only about 0.5 mm. thick, known as the ossific layer, or zone of calcification, in which the real process of ossification takes place—that is, blood vessels grow into it, osteoblasts develop, lime is deposited, and medullary spaces are hollowed out. In healthy bone these two layers are parallel to each other, and are limited by perfectly straight lines. In rachitic bone, on the other hand, they are much enlarged, particularly the proliferative layer, and their naturally sharp boundaries are replaced by an irregular serrated edge, so that the two zones encroach mutually upon each other. Upon microscopical examination, the details of which cannot be given here, we can see most plainly the complete confusion, if we may be permitted to use the expression, into which the growth of the bone has fallen. The proliferation of the cartilage cells has increased beyond all bounds, and the scanty matrix of the cartilage displays a fibrous character. In the bony layer are seen irregularly scattered foci, which are already undergoing incomplete calcification, or else marrow formation, breaking down the cartilage. The latter is due to an invasion of the vessels, which always undergo an active new growth; they permeate the cartilage like lacunar spaces, and are surrounded by a so-called osteoid tissue.

The periosteum presents analogous changes. The innermost osteoblastic layer of the periosteum is thickened; but the newly formed tissue does not become completely calcified, but remains in large part soft and spongy. Finally, an increased absorption of bone takes place inside the bones. The bony partitions disappear, and the cortical layer of bone often becomes much thinner than normal.

These various processes furnish a direct explanation of the macroscopic changes presented by rachitic bones. The proliferative process causes marked swelling of the epiphyses of the long bones, and thickening of the flat bones of the skull. The abnormal softness of the bones is due to the increased absorption of bone, their insufficient calcification, and it in turn causes various deformities, which are, for the most part, very characteristic (*vide infra*). If recovery takes place, the whole bone finally becomes firm; in fact, becomes unusually thick and heavy, but often remains permanently deformed.

The deficient development of rachitic bones can also be recognized upon chemical examination. While normal bones in a dry state contain about sixty-three to sixty-five per cent of lime, rachitic bones have only about twenty to thirty per cent.

Clinical History.—Rickets often begins so insidiously that it can hardly be detected. Attention is not called to the disease until the deformity of the bones becomes very obvious, or it is noticed that the child does not learn to walk as early as other children, or, having already learned, is no longer able to do so. At last the anxiety of the parents is excited; and, on seeking medical advice, they find their fears only too well grounded.

In other cases the appearance of the characteristic changes in the bones is preceded by certain prodromata, which consist partly of catarrhal symptoms of the intestinal canal and the organs of respiration, and especially of certain general symptoms. At night he is restless, screams, bores the back of his head into the pillow, and evinces a tendency to excessive perspiration, so that the pillow is often wet through and through. As a result of the constant rubbing of the head on the pillow, a decided baldness is often perceptible on the back of the head. If the child is laid on his side, he at once becomes quieter. The vasomotor excitability of the vessels of the skin is increased. Slight mechanical irritations produce red streaks and spots. If, before he was attacked by the disease, the child had already made the first attempts to walk, he soon desists. Rachitic children often become pale, limp, and miserable.

The diagnosis of rickets cannot be definitely established until the characteristic changes in the bones have developed. These anomalies vary, of course, in their severity and extent in different cases. We append a list of the most important:

The head is often noticeable for its great size and somewhat square shape ("caput quadratum"), due to the thickening of the parietal and frontal eminences. In proportion to the increased size of the head, the face, which has failed to grow, appears often noticeably small. At the occiput the bones, especially those in the region of the lambdoid suture and the lesser fontanelle, remain compressible like parchment (Elsässer's craniotabes).

The fontanelles remain open till the second or third year of life, and their edges seem soft and yielding. There is often a peculiar change in the shape of the jaws, particularly of the lower jaw. This is not rounded, but angular, being sharply bent in the neighborhood of the canine teeth; so that the incisors stand in a perfectly straight line, besides being somewhat inclined inward. Fleischmann was the first to describe this condition, and referred it to the action of the mylohyoid and masseter muscles upon the soft bone. The upper jaw often appears narrow, while antero-posteriorly lengthened, and prominent like a beak. Dentition in rachitic children is usually tardy, irregular, and tedious. Moreover, the teeth are soft and easily worn away.

The thorax presents, even in the mildest cases, very characteristic and noticeable changes. There is a swelling at the junction of the cartilages with the ribs, which can be felt and seen through the skin, and produces what is called the "rosary of rickets." In severe cases the lateral portions of the thorax are often drawn inward, particularly at the parts which correspond with the insertion of the diaphragm. This change is due mainly to the action of the diaphragm during inspiration upon the abnormally soft and therefore yielding ribs. The changes are greatest when the respiratory efforts, and particularly abdominal respiration, are exaggerated because of bronchitis, pneumonia, or some other disease of the air-passages. In such cases the entrance of air into the lungs is impeded, so that it is possible that the external atmospheric pressure also contributes to produce the deformity of the thorax. Deep hollows may finally be developed on each side of the chest, while the sternum becomes unusually prominent, giving the whole chest that shape which has been termed pigeon breast, or *pectus carinatum*. The lowermost ribs, however, protrude laterally, and project beyond the retracted upper part of the thorax, at times making a decided furrow (Harrison's furrow). When once this deformity has been developed, of course it in turn contributes to render respiration difficult.

The clavicles are sometimes markedly distorted, and may even be partially fractured (*vide infra*). The spinal column is usually unaffected if the child remains quiet in bed; but if it sits up, or is carried about, or tries to walk, the traction and pressure thus exerted often produce curvature of the spinal column (rachitic scoliosis and kyphosis). These deformities may become extreme. Changes in the bones of the pelvis are of no special clinical importance at this period of the patient's life; but in women the consequent shortening of the antero-posterior diameter of the pelvis may, as is well known, prove a great obstacle to childbirth.

The extremities not only present swelling of the epiphyses, but are liable to curvature. This latter change is most marked in the lower limbs, inasmuch as they have to support the weight of the body. The swelling is especially well developed at the lower ends of the bones of the forearm and of the tibia and fibula. The curvature is almost invariably greatest, and therefore most easily recognized, in the tibia, which becomes convex outward, giving the rachitic child its "bowlegs." If a valgus position in the knee and ankle joints makes its appearance, such a condition is called "rachitic knock-knees."

Similar curvature of the femur is less often seen, although it may be obvious enough in severe cases. The same is true of the humerus. The deformity of the lower limbs causes that waddling gait which can be so often seen on the streets of any large city. Sometimes the limbs present a sharp bend, the result of partial fracture. These "green-stick fractures of rickets" are invariably referable to some slight traumatism, and are most often seen in the lower third of the tibia, although sometimes visible in the clavicles, ribs, and bones of the forearm. The infraction usually takes place upon one—generally the concave—side, so that it is often compared to the partial fracture of a quill or an osier rod.

The rachitic bones are probably not spontaneously painful, but are often highly so on pressure. This is a partial explanation of the loud outcries of rachitic children that usually follow firm pressure on the thorax, etc.

Symptoms in Other Parts of the Body.—Apart from the changes in the bones, a rachitic child may seem to be perfectly well. Even the general nutrition may be unimpaired. As a rule, however, rickets is associated with anæmia and impaired nutrition. The muscles, in particular, are small and flabby. The child seems pale, thin, and feeble, and may present swollen lymph-glands and other "scrofulous" symptoms. Sometimes there is a tendency to profuse perspiration, especially from the scalp. Very frequently there is chronic intestinal catarrh, and sometimes there is chronic bronchitis or lobular pneumonia. The abdomen is usually prominent, the liver and spleen are frequently, but not invariably, enlarged. The reader should also be reminded that rachitic children are particularly liable to spasm of the glottis (laryngismus stridulus), tetany, and convulsions, due perhaps to the rachitic lesions of the skull. The mental development of rachitic children is not impeded through this disease.

The feces and urine have been repeatedly subjected to careful chemical investigation in the hope of gaining some information as to the pathogenesis of the disease. The results have been, however, in many respects, contradictory; the most accurate of these investigations have shown that the absorption and excretion of the calcium salts ingested with the food are not very different in healthy and in rachitic children.

The disease almost invariably runs a chronic course. Usually months, or even several years, pass before the process ends. Its termination is to be recognized by a closure of the fontanelles, increase in the length of the bones, and, above all, by the fact that the patient becomes stronger and makes attempts to walk. Unfortunately, many results of the disease persist through life. The legs are crooked, the thorax deformed, the spinal column curved, and the pelvis narrowed. Even in the most favorable cases persons who have once had rickets usually remain somewhat shorter than those who are perfectly healthy, since the longitudinal growth of the bones, especially in the lower extremities, is diminished. In certain cases there is a complete cessation of longitudinal growth, causing the so-called "rachitic dwarfing."

Some authorities describe an "acute rachitis," in which painful swelling of the epiphyses is said to be developed in the course of a few weeks. At the same time the child becomes emaciated, and it may also suffer from diarrhea or ulcerative stomatitis. Recovery takes place in a few months. How far cases of this sort are related to genuine rickets has not yet been determined.

Prognosis.—Rickets does not involve direct danger to life, although many rachitic children fall victims to the attendant intestinal catarrh, or to such complications as catarrhal pneumonia or tuberculosis. The prognosis is, therefore, not unfavorable when the worldly circumstances of the child permit of good care and nourishment. The remote influences of the thoracic, spinal, and pelvic deformities can be readily inferred.

Diagnosis.—The diagnosis of rickets is but seldom difficult if the characteristic changes in the bones exist. In case cranial changes have occurred, we should guard against confounding rickets with hydrocephalus, but we can usually avoid this error. The rachitic child holds its head erect, and is free from mental or other functional nervous disturbances. Infantile myxœdema, hereditary syphilis of the bones, and Barlow's disease may also give rise to errors in diagnosis.

Treatment.—The most experienced pediatrists agree that the first aim in treating most cases of rickets is to improve the general nutrition. It is often possible to bring about recovery simply by means of a proper but not too abundant diet (milk, the yolk of eggs, fresh vegetables, and perhaps meat), good air, sunshine (in the country), and baths (brine, malt, and medicated baths). Digestive disturbances should be corrected by such remedies as hydrochloric acid or tincture of rhubarb; and iron (ferratose, etc.) should be administered if the patient be anæmic.

It is very important that the child should be placed upon a good mattress, and should neither attempt to walk too early, nor be needlessly taken up and carried about. The best way to avoid the development of deformities in the bones is to avoid all such unfavorable mechanical influences.

Attempts have also been made to check the disease by specific remedies. Upon doubtful theoretical grounds, lime has been very frequently prescribed, in the form of phosphate of calcium, of which 15 to 45 gr. may be given in powder several times a day; or in the form of limewater, of which 1 or 2 teaspoonfuls are added to the milk which the child drinks. The benefit of these remedies is seldom very obvious. The ingestion of large amounts of sodium chlorid, lately highly recommended, has likewise failed to prove of practical value. Kassowitz has given a fresh impetus to the employment of phosphorus. To support his belief he brings forward numerous clinical observations, as well as facts obtained from experiment. We may either dissolve the phosphorus in cod-liver oil (0.01 to 100), giving 1 or 2 teaspoonfuls of this solution every day, or we may write for the following mixture, which is more elegant, but is also more apt to spoil:

℞ Phosphori .... gr. ⅙ (gm. 0.01);
Solve in oleum amygdalæ expressum ..... ℥ijss. (10.0 c.c.);
Misce, deinde adde:
Syrupi simplicis, } āā .... ʒj (5.0 c.c.);
Pulv. acaciæ, }
Aquæ destillatæ .... ℥ijss. (80.0 c.c.).
M. Sig.: Two to four teaspoonfuls a day.

We can state from our own experience that the remedy is usually very well borne, and, in fact, often shows its beneficial action after a few weeks, the fontanelles growing smaller and the bones becoming firm. Yet there are cases in which phosphorus does not seem to do any good. But next to dietetic measures, phosphorous therapy is the one which at the present time is to be most recommended.

It may be necessary to resort to orthopedic or surgical treatment in order to correct the deformities of the bones. For details as to these, we must refer to surgical literature.

CHAPTER VI

OSTEOMALACIA

Ætiology and Pathology.—Osteomalacia does not, like rachitis, consist in a disturbance of development. The growing bones are not prevented from ossifying, but, having already undergone normal development and acquired normal firmness, they afterwards become soft. It is mainly a disease of adults, occurring between thirty and fifty years[1] of age, but sometimes earlier. The female sex is noticeably predisposed to the disease, although occasionally it has been observed in men.

The true cause of osteomalacia has not yet been ascertained. It is a remarkable fact that the disease is much more frequent in certain regions than in others. It is very common along the Rhine, and in Westphalia, in eastern Flanders, and northern Italy. This suggests that there is some specific cause for the disease, endemic in certain localities. Among exciting causes, childbearing is certainly the most important, for both the first signs of osteomalacia, and also fresh exacerbations of the disease, usually date from a pregnancy. There is no doubt, however, that occasionally a case of osteomalacia occurs in a woman who has never had children. In such instances the appearance of menstruation seems invariably to have an unfavorable influence upon the disease. In brief, the relations of osteomalacia to the sexual processes are so close that there is some justification for the supposition that osteomalacia is directly dependent for its development upon disturbances of metabolism in the ovaries. Moreover, certain therapeutic experiences (*vida infra*) can well be reconciled with this assumption. It should also be mentioned that unfavorable hygienic surroundings, damp houses, and the like, promote the development of the disease.

The anatomical process of osteomalacia consists of a disappearance of the earthy salts of the bone, which begins interiorly and spreads outward, and causes a corresponding softening of the bony structure. The marrow is at first extremely hyperæmic, and extravasations of blood are not infrequently found here and there. The bony substance surrounding the myeloid spaces and the Haversian canals becomes transformed into a soft fibrous tissue, while the irregularly arranged bone corpuscles either are destroyed or lose their characteristic shape. The softening process gradually extends from the spongy substance outward to the cortex. The central cavity grows larger and larger, so that finally the cortical substance is as thin as paper, and the whole bone like an "inflated and dried coil of intestine." At this stage the original hyperæmia of the marrow has vanished. The marrow acquires a yellow color, and may finally be entirely transformed into a yellow, viscid fluid. The affected bones are now flexible and soft, they can be easily cut, and they are of less specific gravity than normal. The periosteum is also at first thickened and hyperæmic, as if inflamed. When it is removed, the surface of the bone beneath it is found to be rough and uneven. The attendant alterations in the shape of the bones, which can be seen during life, will be mentioned below.

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[1] Rehn maintains that genuine osteomalacia may occur in children; but his statement has not yet been fully corroborated.

Upon chemical examination of the bones in osteomalacia, we naturally find a marked diminution in the proportion of lime salts. It is also stated that lactic acid has been discovered in the bones. This is an interesting fact, as it may be that the acid plays an important chemical part in the process of decalcification.

Clinical History.—Osteomalacia begins very gradually in most cases. Usually the first thing noticed is an ill-defined, deep-seated pain, most often felt in the sacral region, the nape of the neck, and the back añd thighs. The affected parts are also sensitive upon pressure.

The pain is persistent. While it still keeps on, motion becomes gradually impaired. The patient experiences more and more difficulty in walking, partly because of the pain and partly because of muscular weakness. The gait is either uncertain and slow, or characterized by short painful steps, the lower limb and the pelvis being jerked forward as if in one piece. This peculiar gait in which, with every short, hobbling step, the body makes a slight twist inward, is so characteristic that often the disease may be immediately recognized by this alone. In other instances the gait is more waddling. After a shorter or longer time walking becomes absolutely impossible, and the patient is permanently bedridden. Even then severe pain persists in most cases. It is not precisely spontaneous, but is produced by the mere pressure of the mattress or the bedclothes, and similar causes.

In the meanwhile, there has probably been distortion of many of the bones, sufficient to occasion a decided alteration in the appearance of the skeleton. Usually the first change to be noticed is the deformity of the spinal column (see Fig. 107). As a rule, there is kyphosis, less often some other deformity, and the head generally becomes more and more bent forward upon the sternum. As a result, the patient grows decidedly shorter, and this very shortening may be an important factor in diagnosis. The sick woman is apt to remark it, because she has to keep shortening her gown in front. The thorax, also, is apt to be much distorted in severe cases. It is compressed laterally, while the sternum becomes very prominent, and is sharply bent. The change in the shape of the pelvis in osteomalacia

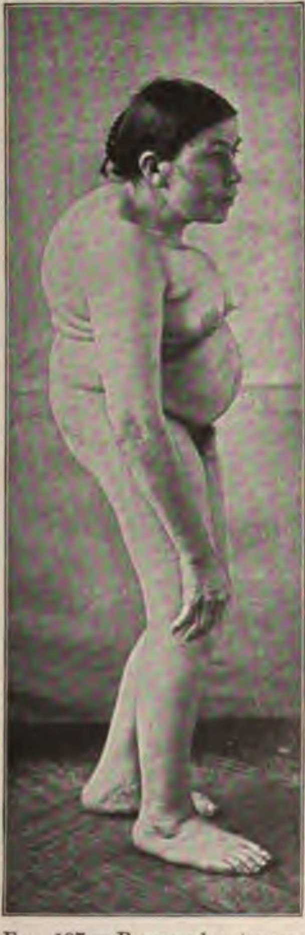

FIG. 107.—Puerperal osteomalacia. (Erlangen Medical Clinic.)

is less obvious externally, but it can be detected on internal examination. It is, of course, of great importance from an obstetrical point of view. The pelvis, like the chest, is compressed laterally, while the symphysis is made to project forward like a beak. The sacrum and its promontory are also pushed forward, and the superior strait thus acquires somewhat of a heart shape. This change in the pelvis is apt to appear very early. It is probably the chief cause of the peculiar gait (*vide supra*). In one case of osteomalacia I found that infraction of the sacrum had given rise to the characteristic symptom-complex of compression of the cauda equina (*vide* page 298).

The extremities are less often distorted, particularly if the patient becomes bedridden at an early period. Manifold changes are, however, possible. Sometimes there is also fracture. In a few reported cases the softness of the bones of the extremities was so extreme that one could bend the limbs at will, like wax, and give them the most extraordinary positions. In cases so far advanced as these, the pain in the bones seems finally to cease. The bones of the head and face seldom undergo noticeable change, but we often find that the teeth are carious or lost. In the muscles, several observers have noticed trembling and fibrillary contractions, also paresis and complete paralysis It is also said that sometimes even a slight irritation of the skin suffices to excite painful contractions of the underlying muscles. These phenomena have not yet been thoroughly investigated.

The general condition of the patient is often unimpaired for a long while, except for the pain and the impairment of motion. The internal organs perform their functions in a normal manner, and the appetite is good. Fever is observed only when the disease is undergoing some temporary exacerbation. With regard to changes in the urine, there have been a good many statements made, but their significance is extremely doubtful. It is said that the amount of phosphoric acid excreted is diminished. With regard to the amount of lime, no definite statement can be made. Lactic acid has been repeatedly detected in the urine, as has also albumen. Concretions of lime have been found in the bladder and the kidneys.

The disease runs a chronic course, occupying seldom less than two or three years, and sometimes even five to ten or even longer. Apparent improvement is not infrequently observed, but this is followed by fresh exacerbations, especially after confinement. The most frequent termination is in death. This results either from general debility, or, more often still, from the dyspnœa caused by the compression of the lungs, or by some such disease as lobular pneumonia. In milder cases, however, particularly if they receive prompt and suitable attention, there may be complete recovery; or in severe cases, even if deformity has occurred, there may be, at any rate, a permanent cessation of the disease.

Diagnosis.—In well-developed cases it is not difficult to recognize the disease, but at first a correct diagnosis is often impossible, unless the endemic frequency of osteomalacia suggests it. In the onset of the disease we may often be led to the false belief that there is incipient disease of the cord or the vertebræ. We ourselves, as well as other observers, have repeatedly seen cases in which women as a sequel to pregnancy, or even apparently spontaneously, have developed paresis in the lower extremities, particularly in the psoas and iliacus muscles, associated with pain and exaggerated tendon reflex, and in which diagnosis at first was very difficult. Not very infrequently the complaint of the patient at the start was regarded as hysterical. Later, the peculiar hobbling and waddling gait ("like a duck") would suggest the possibility of osteomalacia, and this diagnosis would be established by the results of a careful examination of the pelvis.

A rather important fact, which we have substantiated in the last few years, is that there are also lighter forms of osteomalacia which manifest themselves only through pains in the hips and sacrum, and minor disturbances in locomotion, without leading to more serious deformities. We believe that many of the apparently inexplicable pains felt by women in the sacrum, etc., are referable to changes due to osteomalacia. At any rate, this possibility should be kept in mind, and the diagnosis settled by careful examination of the bony pelvis. X-ray examinations of the bones will probably prove particularly valuable in the diagnosis of osteomalacia, although up to the present they are not plentiful enough to be important. Under some circumstances, the diagnosis may be confirmed by the therapeutic results of phosphorous therapy (*q. v.*).

Diagnostic difficulty is often met with in those cases in which paralytic conditions arise before the appearance of demonstrable bony changes due to osteomalacia. Such paralyses can probably be ascribed to an early involvement of the muscles in the pathological process. The possibility of incipient osteomalacia should, in such cases, too, be most particularly borne in mind.

Treatment.—As has been already implied, therapeutic efforts have thus far proved in severe cases almost unavailing in this disease.

In incipient and light forms of osteomalacia, however, and sometimes even in the more severe cases, phosphorus is, in our opinion, undoubtedly a specific remedy. In a whole series of light and medium cases of this disease we have had very satisfactory results from the long-continued employment of phosphorus in an oily solution (0.01 parts of phosphorus in 100 of cod-liver or olive oil), or, instead of the oily solution, the phosphorus may be administered in keratin-coated capsules. The use of phosphorus should be supplemented by hygienic and dietetic remedies, salt baths, CO_2 baths, country air, and wholesome nourishment, etc.

VII. CONSTITUTIONAL DISEASES

ANOMALIES OF THE BLOOD AND TISSUE METABOLISM

CHAPTER I

GENERAL PRELIMINARY REMARKS ON THE PATHOLOGY OF DISEASES OF THE BLOOD. (*Cf.* PLATE V.)

IN few divisions of pathology have our points of view and knowledge been so broadened and deepened within the past decade as in the study of the diseases of the blood and of the hemopoietic organs. Furthermore, the pathological examinations have also acted unusually stimulatingly and fruitfully on the research into normal conditions, especially the anatomical and histogenetic, to an extent equaled only in the field of neurology. Through this there has developed such a close relationship between the normal and pathological findings that, for a better understanding of the latter, a short review of the general morphology of the blood is absolutely necessary. To be sure, this field is an unusually difficult one, and there still exist wide differences of opinion concerning many isolated questions, even among the most competent investigators. The following *résumé* can, therefore, take into consideration only the most important and almost universally accepted details:[1]

The starting point of all the newer investigations in the domain of blood diseases is the discovery of leukæmia by Virchow in 1845. Virchow and Max Schultze also first taught us to recognize the differences between the separate forms of the white blood cells. The greatest stimulus for further progress was the discovery of Neumann, in Königsberg, of the hemopoietic function of the bone marrow in 1870. Herewith, this hitherto lightly regarded organ suddenly became the center of all further investigation. An unusually great advance was then registered by the fundamental and creative work of Paul Ehrlich upon which, even now, the entire structure of the pathological morphology of the blood for the most part rests.

As is well known, we distinguish among the formed elements of the blood, the red blood corpuscles (erythrocytes), the white blood corpuscles (leucocytes), and the blood plaques.

1. **Red Blood Corpuscles.**—The red blood corpuscles (erythrocytes, normocytes, Plate V, Fig. 1–4, *a*), in the embryonic development, spring from the

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[1] As an introduction into a more accurate study of the diseases of the blood, I recommend, above all, the excellent book of Otto Naegeli, "Blutkrankeiten und Blutdiagnostik," Leipsic, 1907; also, Türk's "Vorlesungen über Klinische Hämatologie," Vienna, 1904; E. Grawitz, "Klinische Pathologie des Blutes," Leipsic, 1906; Ehrlich, Lazurus u. Naegeli, "Die Anämie," Vienna, 1909.

cells of the primitive vascular nucleus, whose peripheral cells develop into endothelium, and whose central cells become the so-called erythroblasts, and later the erythrocytes. The formation of the red blood cells far precedes that of the leucocytes. This is a most important fact, as it renders, *a priori*, highly improbable the development of erythrocytes from leucocytes, which was formerly assumed, and which is even now not entirely discredited. With the progressive development of the embryo, the blood formation localizes itself more and more in a few definite organs. It is, in the first place, particularly marked in the liver, then in the spleen, and after the third month of embryonic life it begins in the bone marrow. In post-embryonic life the blood formation in the liver and spleen ceases entirely. This function is discharged exclusively by the bone marrow, both the long bones and the short, flat bones being active in children. In later life generally the latter only (sternum, ribs, vertebral bodies, base of skull) still possess "red" blood-forming marrow. All the ordinary, nonnucleated erythrocytes of the circulating blood have their origin in the nucleated red blood corpuscles, which are normally always found in the bone marrow, but not in the blood into which only the completed elements enter. The nucleated erythrocytes are therefore termed normoblasts (Plate V, Fig. 2, *g*; 3, *b*; 4, *b*). They have even in this stage a distinct hemoglobin content, and usually a rounded nucleus with marked staining properties. We distinguish the megaloblasts from the normoblasts (Fig. 2, *i*). They are decidedly larger than the normoblasts, and have a much paler protoplasm, poor in hemoglobin, with a large nucleus, with far weaker staining properties. Under normal conditions the megaloblasts appear only isolated in the bone marrow. They are looked upon as an older form of cell formation, from the ontologic (and phylogenetic) standpoint. The appearance of numerous megaloblasts in certain diseases of the blood is therefore interpreted by Ehrlich as a sort of reversion to embryonic conditions. The transition of the nucleated normoblasts into nonnucleated erythrocytes takes place in all probability by so-called karyolosis—i. e., through chemical intracellular solution of the nuclear substance. The expulsion of the nucleus, assumed by many authors, appears to occur only as a pathologic process dependent upon certain physical changes.

The number of red corpuscles amounts to about 5,000,000 per c.mm. in the adult male, and 4,500,000 in the female, under normal conditions. The hemoglobin content of the blood in disease does not by any means always correspond to the number of erythrocytes, so that obviously quite large variations of the hemoglobin content of the erythrocytes occur. The hemoglobin content of the blood is generally determined by the calorimetric method (hemoglobinometer of Sahli-Gowers, hemometer of Sahli), and the normal hemoglobin content is fixed at one hundred per cent. The hemoglobin content, for example, of fifty per cent signifies therefore a diminution of the hemoglobin to one half. By comparison of the erythrocyte count with the hemoglobin content, one can form an opinion of the hemoglobin content of the individual red blood corpuscles (the so-called color index). For example, if the hemoglobin content of the blood reaches fifty per cent, while the number of red blood corpuscles is only slightly diminished, let us say, to 4,000,000, this signifies a marked reduction in the hemoglobin content of the red blood corpuscles; while, *vice versa*, a hemoglobin content of fifty per cent, with a diminution of the red blood corpuscles to 1,500,000, indicates an increased hemoglobin content of the individual erythrocytes.

Besides the number and the hemoglobin content of the red blood corpuscles, one must also take into consideration changes in their size and form. In the normal blood, all erythrocytes are practically of the same size, their diameter being about 7.5 μ, with normal variations between 6 and 9 μ. In diseases of the blood one often notices at once a marked irregularity in the size of the red blood corpuscles (anisocytosis). One finds besides the normal sized normocytes, abnormally small microcytes (2, *d*) and abnormally large megalocytes (2, *c*). The microcytes are probably poorly nourished, underdeveloped cells; in part, apparently, also segmentation products. The megalocytes are due partly to swelling, and are then paler than the normal erythrocytes; or they are really hypertrophic red blood corpuscles, with an increased amount of hemoglobin. They then correspond to the normal embryonic megalocytes, and, to a certain extent, are again a reversion to the embryonic condition. On the other hand, we believe, their formation might be regarded as a compensatory process, analogous to the occurrence of hypertrophic muscle fibers in many forms of progressive muscular atrophy. If the red blood corpuscles show irregular forms, pear-shaped, anvil-shaped, etc. (2, *b*), instead of their normal rounded shapes, we speak of poikilocytosis. This depends upon some abnormal lack of resistance of the red blood corpuscles toward physical and mechanical influences. A portion of the poikilocytes are also, perhaps, products of segmentation (schizocytes).

Finally, we have to mention certain variations from the normal staining properties which are clearly referable to changed chemical conditions. Normal red blood corpuscles stain with acid dyes (eosin), but not at all with basic ones (methylene blue). In pathological conditions the blood corpuscles sometimes also stain with basic dyes, thus assuming, on staining with eosin methylene blue, a faded violet shade (so-called polychromasia or polychromatophilic degeneration, Fig. 2, *f*). This behavior is due partly to certain degenerative changes in the red corpuscles, but in part is probably also to be viewed as the appearance of young and embryonic cells, since polychromatic cells are always found in the bone marrow and in embryonic blood. Their occurrence in the blood would, accordingly, at least in part, be analogous to the appearance of nucleated red blood corpuscles. Lastly, we mention the so-called basophilic granulation of the red blood corpuscles—i. e., the appearance in the red blood cells of fine granules which stain deeply with basic dyes. A special diagnostic significance has been ascribed to this phenomenon from the fact that it is almost invariably observed in chronic lead poisoning, although also seen in various anæmic states, especially in pernicious anæmia. The origin of the granules has not been fully explained. Probably we have not a toxic degeneration of mature erythrocytes, but the product of an abnormal embryonic reaction of the bone marrow, corresponding to the appearance of polychromatophilic cells (O. Naegeli).

2. **Leucocytes.**—The leucocytes (white blood corpuscles) must be divided into two large groups, according to their myeloid or lymphatic origin.

In adults the myeloid tissue system, as already mentioned, is chiefly localized in the bone marrow; during fetal life the liver and spleen (pulpa) also contain myeloid tissue, and accordingly also have leucopoietic functions, which may reappear under pathological conditions. The lymphatic tissue system arises autogenetically later than the myeloid. It occurs, first of all, principally in the thymus, then in the lymph-glands and in the numerous other lymphatic apparatuses (follicles of the spleen, tonsils, follicles of the mucous membrane, etc.). The cells generated from these two tissue systems require a separate discussion:

I. To the myeloid cells pertains, first of all, the major portion of the ordinary white cells, which always occur in the blood, and which are at present termed the neutrophile polynuclear (polymorphonuclear) leucocytes. Their number is normally about 5,000 to 7,000 in a c.mm.—i. e., about sixty-five to seventy per cent of all white cells. Their protoplasm is characterized by its neutrophile granulations—i. e., staining with neutral aniline dyes. The nuclei appear singly in a peculiar form, or else in increased number (two to five; Fig. 1, *b;* Fig. 2, *c;* and others). These cells possess the faculty of active amœboid motion; they are able to penetrate through the capillary walls, and therefore go to form the overwhelming majority of "pus corpuscles." They produce proteolytic ferments (purulent liquefaction of the tissues), and act as phagocytes. The origin of the neutrophile leucocytes from the bone marrow has been shown with certainty. They proceed from the so-called myelocytes, which are always found physiologically in the bone marrow, but under pathological conditions also enter the blood. The myelocytes are considerably larger than the neutrophile leucocytes (diameter, 15 to 20 μ). They have a large, circular, pale nucleus without nucleolus, and an abundant fine granulated protoplasm (Fig. 4, *e*). A still earlier stage of the myelocytes is formed by the myeloblasts—i. e., large cells with one large nucleus, but nongranulated protoplasm. These are always present in bone marrow, but under given circumstances—above all in myelogenous leukæmia—may appear in the blood in large numbers. While, as just stated, the chief forms that develop from the myeloblasts are neutrophile myelocytes, and from these again, neutrophile leucocytes, two other varieties of myelocytes also emanate from the myeloblasts, though in smaller numbers. These are characterized by eosinophile (acidophile) and basophile granulation of their protoplasm. From these two forms arise the eosinophile leucocytes and the basophile leucocytes, or the so-called "mast cells" (Fig. 4, *g. h,* and *i*), all of which probably have a definite, but as yet wholly unknown, physiological importance. These two forms of leucocytes are always present in normal blood, though in small numbers. The eosinophiles constitute two to four per cent of all leucocytes; the basophile mast cells only about one half per cent. If we assume the total number of white cells as 10,000, approximately 7,000 cells are neutrophiles, 200 eosinophiles, and at most 100 basophiles. The remainder of about 2,700 is made up of lymphocytes (*vide infra*). Each of the three preceding kinds of cells must be regarded as an individual biological form, and not as the expression of a transient physiological functional condition. The various forms of leucocytes therefore never merge into each other. Their granules are to be considered as products of the specific cell metabolism, or of the specific cell growth.

II. Entirely different from the myeloid system is the lymphatic system, whose cell products are called lymphocytes. The ordinary lymphocyte of normal blood is about the size of a red blood corpuscle, and has a round nucleus that stains deeply with basic dyes, and is surrounded only by a narrow, nongranulated fringe of protoplasm (Fig. 1, *c;* 3, *d*). The lymphocytes have but little motility, and generate no proteolytic or oxidizing ferments. They are formed in the lymph-glands and in the follicles of the spleen. The spleen, in a sense, is the lymphatic gland of the blood. But lymphatic tissue is also found scattered in other organs, and to a slight extent, probably, also in bone marrow. The lymphatic tissue consists almost entirely of so-called lymph follicles, in whose center (germinal center) the genesis of the cells occurs. The initial stage of the small blood lymphocytes is formed by the so-called large lymphocytes, which also have a large circular nucleus and thin protoplasmic border. Under pathological conditions (lymphatic leukæmia) they appear in abundant numbers in the blood, just as the myelocytes do in myelogenic leukæmia.

In connection with all the cell forms thus far discussed, we have to consider two others whose importance is still much debated. First of all, the so-called large mononuclear cells and "transition forms," which often used to be confused with the large mononuclear lymphocytes, but must, in all probability, be separated from them, and classed with the myeloid system. Ehrlich regards them as an intermediate stage between the nongranulated myeloblasts and the neutrophiles. The large "mononuclears" also appear in normal blood, about 200 to 400 to the c.mm. We cannot here enter into their minute histological peculiarities. Lastly, we would also mention the so-called plasma cells—large cells with very strongly basophilic, honeycomb-like protoplasm and round nucleus. They occur only pathologically in the blood, and rather more frequently in the tissues of inflammatory processes. Their origin is uncertain. Apparently it may be myeloid as well as lymphatic. They have no special diagnostic significance.

As the most important fact of all these considerations, I would emphasize that the blood is the meeting place of cells from three different tissue systems, the erythropoietic, the myeloid-leucopoietic, and the lymphatic. An exact examination of the blood cells gives us direct information of any variation in the function of these tissue systems by showing either a diminished, an increased, or a qualitatively altered activity thereof. In conjunction with other symptoms, this functional diagnosis also often permits certain conclusions regarding the anatomical condition of the tissues in question, or of the organs formed from them (bone marrow, lymph-glands, spleen, etc.). All these propositions will find further elucidation in subsequent chapters.

3. **Blood Plaques.**—The blood plaques are small, colorless bodies (2 to 3 μ in diameter) that are present in every specimen of blood. They have the tendency rapidly to agglutinate in small clumps. They possess no real nucleus, but are easily stained with dyes. Their number in each c.mm. is about 200,000 to 300,000. They appear to have a certain amœboid movement. In the past they were frequently regarded as degenerative products of the leucocytes, while nowadays they are regarded rather as independent bodies. The important rôle they play in fibrin coagulation has been proved with certainty. They contain the "thrombogen" necessary to the formation of fibrin ferment (Morawitz).

CHAPTER II

ANÆMIA AND CHLOROSIS

General Preliminary Remarks as to the Classification and Ætiology of the Various Forms of Anæmia.—The word "anæmia" (or, more strictly, oligæmia) might properly be taken to signify diminution of the total volume of the blood, such as, for example, is directly brought about by a severe hemorrhage. Usually, however, the word is employed to signify not so much diminution in quantity as deterioration in quality, with special regard to the number and character of the red blood globules. The total volume of the blood is not liable to nearly so great variations as is the number of its most important constituents—the red corpuscles—inasmuch as the total volume is dependent merely upon the amount of watery constituents, and even after large hemorrhages the water is quite rapidly replaced by absorption. This is undoubtedly the case in most instances of sudden loss of blood. Even in chronic anæmia there is usually no reason to assume that the total amount of blood is diminished, although it may be when there is general emaciation, or diminished supply of liquids (persistent vomiting, dysphagia), or large watery discharges (as in cholera). The essential element in anæmia is, therefore, a diminution in the number of the red blood corpuscles, or so-called oligocythæmia. Changes in the character of the red blood corpuscles are not taken into consideration here. On the other hand, however, we shall see later that, even in spite of a normal number of red blood corpuscles, their abnormal condition, particularly their poverty in hemoglobin, may lead to well-marked "anæmic" conditions.

The circumstances under which anæmia is observed are manifold. They admit, however, of our distinguishing two great classes of anæmia—primary and secondary. Primary anæmia is developed as an apparently primary and idiopathic disease in persons previously healthy, while secondary anæmia is merely a symptom of some already existing disease. However simple this theoretical distinction, yet in actual practice it is often quite difficult to determine whether the particular case before us should be regarded as primary or secondary. A secondary anæmia may occur where the true primary cause cannot be at all readily determined. There are, nevertheless, quite a large number of cases which would seem to deserve the name of primary or essential, in which we feel compelled to assume that some pathogenic influence acts directly upon the blood and the hematopoietic processes.

In the first place, we would class as primary, cases which may best be described as "simple constitutional anæmia." These often stand close to the borderland between health and disease. There are not a few individuals who present a striking pallor for a large part, if not all, of their lives.

This sort of facial pallor by no means always necessarily indicates true anæmia. It may be a result of insufficient development of the cutaneous capillaries, or a thickness of the epidermis, etc. In such cases, nevertheless, careful examination will sometimes reveal anæmia, though usually not very considerable. If these persons feel well and vigorous we scarcely have a right to regard the existent anæmia (usually moderate) as an actual disease. Sometimes, however, such individuals do betray some diminution of energy, are easily fatigued, and are subject to headache. We may then regard the conditions as certainly pathological. In many instances the cause of this simple anæmia is found in the general hygienic surroundings of the patient, for such cases are most often met with among the poorer classes. Deficient nutrition, bad air, unhealthy occupation in factories or the like, not only affect the general health, but more especially interfere with the processes of normal blood-making. Other cases of constitutional anæmia, apparently primary, occur in individuals who are entirely beyond the reach of such influences as have just been mentioned, in whom the anæmia has developed and persists despite the best of food and air. Here we are forced to the conclusion that the organs engaged in the manufacture of the blood are in some way prevented from performing their proper functions. The trouble often seems to be congenital, for such individuals may present the symptoms of anæmia in their earliest infancy. There are persons who have always been pale and feeble. Or, again, anæmia does not develop until later on, in which case it not infrequently associates itself with certain phases of physiological development, as when growth is particularly rapid, or when adolescence occurs. Virchow has directed attention to another factor, which he regards as potent in many of these cases of congenital anæmia. He has found that the arteries may be congenitally small, or that the whole arterial system may be imperfectly developed. The condition may be associated with a congenitally weak and small heart. The importance of this factor has not yet been fully determined. Possibly the condition of the circulatory system just mentioned may be the result rather than the cause of the anæmia.

A second division of primary anæmia includes cases which present a far more definite and distinct group of symptoms. They quite often appear in persons previously healthy, last for a certain length of time, and then end in complete recovery. The typical form of this variety is chlorosis (χλωρὸς = greenish-yellow), or "green sickness." This well-known disease is especially frequent in young girls fourteen to twenty years of age—that is, at puberty. It often comes on quite rapidly without any ascertainable cause. There are not infrequently predisposing influences in the outward circumstances of the patient. Thus it is promoted by an unhealthy sedentary mode of life, as in seamstresses; bad air, as in factory operatives; lack of sunlight (waitresses in poorly lighted places; mental and physical overexertion, as in teachers, governesses, and students; and, finally, by mental influences (excitement, etc.). It is, nevertheless, true that chlorosis also appears in girls who have lived under the most favorable hygienic conditions possible. Sometimes the disease seems to be merely a temporary exacerbation of a simple constitutional anæmia which has existed a long time; but it may also appear in young women who have been previously healthy, and even robust.

The true cause of chlorosis is unknown. Perhaps the interesting investigations of Bunge with regard to the amount of iron in milk and in newborn animals afford at least a starting-point for some future theory as to chlorosis. Bunge found that the constituents of the ash of milk, and the constituents of the total ashes of young suckling animals, correspond precisely in their proportions. The suckling animal, therefore, receives with his mother's milk all the salts that are needed for his body, in a proper amount—the single exception being the iron compounds. The amount of iron in milk is very small compared with the amount of iron in the suckling. Indeed, the proportion of iron in the newborn animal is decidedly large, but it diminishes gradually as the creature develops. This indicates that the animal comes into the world with a supply of iron which can originate only in the maternal organism; and in the first part of his life, so long as he has milk for his exclusive nourishment, he draws upon this supply. If, therefore, we consider that it is a function of the female organism in some way to store up iron for the next generation, there might be some disturbance of this function, either an imperfect assimilation or an abnormal conversion of the assimilated iron, and this might very likely rob the blood of hemoglobin. It must be confessed that as yet we have no thorough understanding at all of the internal processes of metabolism which relate to the matter. Still, the facts and conditions which we have mentioned explain to a certain degree the fact that genuine chlorosis is found exclusively in the female sex and, indeed, precisely at the time of puberty—i. e., the time of preparation for the sexual life. Perhaps, also, the fact that disturbances of menstruation are so frequent in association with chlorosis is connected with the conditions above mentioned, but it is also possible that the menstrual disturbances may be a result of chlorosis. We cannot, however, ignore the possibility that abnormal internal secretory processes of the ovaries may have a harmful influence on the production of blood by the bone marrow.

A third form of primary essential anæmia is the so-called pernicious anæmia. This, likewise, appears independently of other diseases, and is characterized by its steady progress, as a rule leading finally to death. We do not regard pernicious anæmia as a very severe form of chlorosis, but as an entirely separate disease. It is distinguished from chlorosis not merely by the much more definite morphological changes in the blood, but still more by the circumstance that it is not confined to women, but appears also in men, both in early and advanced life. Cases of "grave essential anæmia" also have many points in common with certain other diseases, such as pseudo-leukæmia and splenic anæmia. These will be discussed later.

The forms of secondary anæmia offer a contrast to the forms of primary or essential anæmia just described in the much greater number of their causes. Under this head come cases of anæmia which do not occur idiopathically, but as a result of other abnormal processes. The simplest variety is anæmia from hemorrhage. This is produced by profuse loss of blood, whether from the stomach, lungs, uterus, intestines, kidneys, or some wound. Repeated small hemorrhages finally produce the same result as a single large one. Thus, the most profound anæmia may be observed where there is a very frequent epistaxis (hemorrhagic diathesis, contracted kidney), or where cancer of the womb or of the stomach gives rise to a constant oozing of blood. The direct loss of blood always leads to an equivalent diminution of the red blood corpuscles and the hemoglobin. The restoration of the blood is possible only through an increased activity of the bone marrow, as a result of which we frequently find an abundant production of normoblasts, polychromatophilic cells and an increased number of leucocytes.

Besides these directly explainable forms of anæmia, there are numerous secondary anæmias in which there is no possibility of such direct loss of blood.

If we analyze these cases more closely it seems to us that we can distinguish above all two groups of secondary anæmia. In one group the anæmia is a symptom of impaired nutrition. This is seen in almost every severe disease, acute or chronic, and is usually associated with more or less emaciation and loss of strength. The bad appetite, the lack of fresh air and exercise, and perhaps an impairment of digestion, or fever, or some abnormal drain upon the system, as in suppuration—these injure the entire body. It is not surprising that the blood shares in the universal misfortune. This is why most chronic invalids seem pale, particularly if they suffer from disease of the stomach, kidneys, chest, or nervous system. In the second class the anæmia is secondary to some other disease, but assumes especial prominence as a symptom, independently of any general impairment of nutrition. Of course, it is often associated with emaciation, but nevertheless its extraordinary intensity offers a striking contrast to the condition of the rest of the body. This "specific secondary anæmia" must, like essential anæmia, be due to a special lesion of the blood itself, and is, therefore, to be regarded as in a certain sense a special complication or localization of the primary disease. General malnutrition never produces anæmia of this grade directly. This fact is illustrated in cicatricial stenosis of the œsophagus. Here the ingestion of food is very greatly, if not completely, hindered, and there is the greatest emaciation, with a subnormal temperature and slow pulse, and still the external appearances as well as the blood picture of genuine anæmia are wanting.

The exact mode of origin of specific secondary anæmia is often obscure. We have already reported, in a preceding chapter, a very instructive example. In cancer of the stomach we find usually emaciation and pallor. This is natural enough; but sometimes the carcinoma is complicated by an extraordinarily profound anæmia, comparable only to the pernicious variety. In one such case we found, at the autopsy, an extensive secondary carcinosis of the bone marrow. Here, therefore, the anæmia certainly was not the result merely of the general impairment of nutrition occasioned by the gastric carcinoma, but of the disease of the marrow, which tissue undoubtedly plays an important part in the manufacture of the blood.

Some cases of genuine secondary anæmia are sometimes observed to develop after certain acute infectious diseases. For example, there may be great anæmia after typhoid fever, or less often after acute articular rheumatism. A peculiar form of anæmia is often observed during the secondary stage of syphilis, although nutrition seems to be otherwise well maintained. This is known as "syphilitic chlorosis." Tuberculosis, chronic malarial poisoning, chronic septic infection, and other forms of chronic poisoning (lead), as well as amyloid disease, renal disease, etc., may also be attended by anæmia of such intensity as to justify the conclusion that there is some special disturbance of secondary origin affecting the manufacture of the blood or the blood itself. Of great importance is the striking anæmia which occurs in association with many intestinal parasites (see Vol. I, page 628 *et seq.*), sometimes as a result of actual loss of blood, but probably more often because of the action upon the blood of certain poisons generated by the entozoa.

We shall now proceed to describe the symptoms directly dependent upon the anæmia, and therefore common to all its forms. Upon this will follow a sketch of chlorosis. A special chapter is assigned to the grave form of essential anæmia known as progressive pernicious anæmia; and in the same connection will be set forth the little that is known with regard to the relations of anæmia to pathological changes in the hematopoietic organs.

Symptomatology of Anæmia.—The first symptom which attracts the attention of the physician in any case of anæmia is the altered appearance, the pallor of the skin and visible mucous membranes. This is especially striking in the face, but it is sufficiently evident everywhere. Special value is usually assigned to pallor of the mucous membranes—for example, of the lips and conjunctiva, inasmuch as their color is not liable to be interfered with by pigmentation or opacity of the epidermis. The degree of pallor, of course, varies greatly. The whole body may present a waxy appearance.. A pallor of this kind implies, of course, a very considerable diminution in the amount of coloring matter in the blood—that is, of hemoglobin. The diminution of hemoglobin in simple anæmia is usually a direct result of diminution in the number of red blood corpuscles, but, as already mentioned, there may be a diminution in the amount of hemoglobin contained in each blood corpuscle (low color index).

Besides the anæmic complexion, we observe in all cases a series of symptoms whose ultimate cause is probably chiefly to be sought in a weakening of the normal processes of innervation, due to lack of arterial blood. Here belongs first the general feebleness, the comparatively rapid exhaustion of the muscles under voluntary control, and the associated feeling of languor. In the severest anæmia (e. g., after great loss of blood) the motor weakness may be so considerable that the patient can neither walk nor stand. But even in milder degrees of anæmia there is evident feebleness of body in greater or less degree.

A corresponding diminution in the processes of innervation is sometimes found to affect the senses and the mind. This is shown especially in the general mental weakness, the inability to think deeply, the constant sense of fatigue and sleepiness. In severe anæmia there are often attacks of faintness, and even of actual syncope, because of cerebral anæmia. Patients very often complain of impaired vision, specks before the eyes, pain in the eyes, etc. Occasionally, especially after very severe hemorrhages (e. g., severe hematemesis), there is complete amaurosis. Examination with the ophthalmoscope shows nothing abnormal beyond the anæmia of the retina. Impairment of hearing is exceptional; on the other hand, there is often great annoyance from marked tinnitus aurium, which, of course, is due to the irritated condition of the auditory nerve, or perhaps also of the auditory center.

The processes of secretion are much less affected. It is true that there is often dryness of the tongue and mouth, but this is mainly subsequent to the loss of large amounts of blood, and probably, therefore, is occasioned less by a diminution in the activity of the mucous and salivary glands than by the desiccation of the tissues. As relates to other secretory functions, the only one which has been carefully investigated is the formation of gastric juice. The earlier experiments on animals caused the opinion to be held for a long while that in anæmia the proportion of hydrochloric acid in the gastric juice was diminished. Numerous more recent experiments, however, have shown that this assumption is subject to exceptions. Although the amount of hydrochloric acid in the gastric juice may be small in anæmia and chlorosis, and although there may be complete absence of free hydrochloric acid in the gastric juice, yet, on the other hand, it is a very frequent experience to find a fully normal or even excessive amount of hydrochloric acid; nor are the motor functions of the stomach, as a rule, disturbed; but it may be that the constipation which is so often present in anæmia is, at least in part, connected with an impairment of the energy of intestinal peristalsis.

The symptoms thus far described are referable partly to diminished functional activity. On the other hand, anæmia gives rise to certain symptoms of irritation in the nervous system. These are, in all probability, expressive of the irritation excited in certain portions of the nervous system by the products of abnormal metabolism, perhaps due to incomplete oxidation. Some of these symptoms, such as the annoying tinnitus, the specks before the eyes, and the vertigo, have been already mentioned. We should also note especially eructations and vomiting as anæmic symptoms; these are in most cases of central origin, and in severe anæmia they may be very annoying. In the same way may be explained the occasional occurrence of annoying hiccough, the frequent spasmodic yawning, and similar symptoms. Anæmic headache may be very severe. It is usually referred either to the entire head or to the forehead; there is a painful feeling of pressure, which may attain great severity.

Other important evidences of irritation are to be found in the behavior of the pulse and the respiration. The changes here are apparently in part of a compensatory character. The pulse is accelerated in most cases of any severity, reaching 80 to 100 beats per minute, or even more. It is also very excitable, so that comparatively slight external influences suffice to increase its rapidity for the time being. This increase in frequency would not, of course, necessitate an increase of intravascular tension, or of the rapidity of the circulation; but it may exert a favorable influence in this direction, and so be compensatory. Respiration also is usually accelerated in anæmia. In cases of great severity the breathing may be so deep and noisy as to justify the term "anæmic dyspnœa." This is the direct expression of the body's hunger for oxygen. It is obvious that this increase in the number of respirations favors, to a certain extent, the absorption of that gas.

There are still other symptoms referable to the circulatory system. It has been already stated that the total volume of the blood is not diminished in anæmia unless, of course, there has just been an actual hemorrhage. This explains why the pulse in anæmia is often comparatively full and strong. Quite frequently there is a peculiar quickness of the pulse. This seems to be due to a vigorous cardiac systole, in conjunction with a low intra-arterial tension. Akin to these phenomena is the fact that in severe anæmia it is not exceptional to hear a distinct pistol-shot sound in the femoral artery, or, in rare cases, even a distinct double sound, just as in aortic insufficiency.

It has long been known that anæmia may produce functional cardiac murmurs, often called "anæmic murmurs." Their mode of origin is not yet fully explained. They are usually heard loudest over the base of the heart, in the neighborhood of the pulmonary valves, and at the apex of the heart. As a rule, they are purely systolic in time, but we certainly heard in one case of pernicious anæmia a loud diastolic murmur of anæmic origin. The murmurs are of a blowing character. Sometimes they are so rough as to stimulate pericardial friction murmurs; it has therefore been suggested that many anæmic murmurs are actually due to the rubbing upon each other of the abnormally dry folds of the pericardial sac. Another explanation of their occurrence is, that the movements of the cardiac valves are interfered with, as a result, perhaps, of fatty degeneration of the myocardium (*vide infra*). It is also possible that there may be sometimes relative insufficiency of the valves—e. g., due to dilatation of the heart or to imperfect action of the papillary muscles. Still, the question has not yet been settled whether genuine dilatation of the heart is at all frequent in anæmia. Increase in the cardiac dullness is observed, but it is due mainly to retraction of the lungs.

Murmurs in the large veins of the neck are very often heard in anæmia, either with or without cardiac murmurs. They are often called *bruit de diable*. No less an authority than A. Weil has maintained that murmurs in the jugular veins are just as frequent in healthy persons as in the anæmic, but our experience compels us to differ from this view. We believe that the loud venous murmurs are more frequent in the anæmic than in other persons, so that they possess a certain diagnostic value.

The processes of metabolism in profound anæmia are of great interest, but, unfortunately, they have not yet been thoroughly investigated. *A priori*, we should expect that the respiratory interchange of gases would be diminished as a result of the impoverishment of the blood in respect to its hemoglobin contents, but this, according to the concurring results of numerous examinations, is by no means the case. On the contrary, even in profound anæmias, the consumption of O and the production of CO_2 retain their normal ratio. Therefore, we must assume that the body has means of adjustment (acceleration of respiration, of the pulse, etc.) which make it possible to take in sufficient O. The decomposition of albumen displays remarkable changes. As I demonstrated years ago, and as has since been repeatedly confirmed, in severe anæmias, we occasionally, if not always, find a remarkably large nitrogen excretion in the urine, in proportion to the albumen taken—i. e., an increased destruction of albumen.

The urine generally is rather pale, its total quantity sometimes relatively large (3 to 4 pints [1,500 to 2,000 c.c.]), its specific gravity rather high (1.015 to 1.021), which last fact may sometimes be referred to the rather large proportion of urea which is present. Albuminuria is exceptional in simple anæmia.

The fatty degeneration of the internal organs (heart, kidneys, blood vessels), which occurs in almost every case of marked anæmia, is of clinical importance, but its origin has not as yet been explained. It has already been suggested that the fatty degeneration of the heart may be the cause of certain irregularities in its functional activity; but it should be said that this is not invariably the case, for often the heart exhibits a surprising energy despite marked fatty degeneration of its muscular tissue. The corresponding changes in the walls of the blood vessels are, however, of great importance, as they frequently occasion disturbance, above all by hemorrhage. In many instances (for example, in leukæmia, *vide infra*) an actual hemorrhagic diathesis is developed; but we shall find later (see the chapter on Pernicious Anæmia) that other factors may also play a part in producing capillary hemorrhages in severe anæmia. There is strong evidence in many cases of an abnormal permeability of the vascular walls. From this results the mild grade of œdema frequently seen in anæmic patients, although in some few cases this œdema may be ascribed to passive congestion, resulting from the cardiac debility (*vide supra*). The increased permeability of the renal blood vessels is sometimes shown by polyuria.

The bodily temperature is very often affected in anæmia. An "anæmic fever" is very frequently associated with pernicious anæmia, and even with cases of profound secondary anæmia, as after a large hemorrhage from the stomach. There are irregular elevations of temperature, usually occurring in the evening, and attaining 101° to 102° F. (38.5° to 3° C.), or even still higher figures. This fever is not due to inflammatory organic changes, but, in all probability, is rather the result of the presence of toxic substances in the blood.

Clinical History of Chlorosis.—Chlorosis, or "green sickness," is a definite form of primary essential anæmia, which appears exclusively in the female sex and in the years of adolescence, mainly from the fifteenth to about the twentieth year of life. The disease either may appear quite suddenly in a previously healthy girl, and in such a case may completely disappear again after a few weeks or months, or the entire course may be a chronic one without sharp limits, so that the condition approaches more nearly to a constitutional anæmia. In many cases, also, it would be proper to say that there are recurrences of chlorosis, for severe attacks of the disease may be seen more than once in the same girl. As a rule, there is no definite cause to be found for the appearance of chlorosis, except that sometimes it seems to be promoted by an unhygienic mode of life, too many social engagements, or long-continued mental excitement.

The various symptoms of chlorosis are almost all direct results of the anæmia, so that they have already been described. Their intensity and variety are, however, very different in different cases. There are mild cases where the patient can hardly be called ill. She feels perfectly well, but is "a little pale." From these cases there is a gradual and unbroken transition to the other extreme of severity.

A constant and essential symptom is the greater or less pallor of the face as well as of the rest of the surface of the body, and of the mucous membranes so far as visible. This pallor sometimes really has a greenish tint. There is almost invariably general languor; the patient is easily fatigued, and has neither the desire nor the ability to make any great bodily or mental exertion. There is also a tendency to headache and vertigo. Chlorotic patients often complain of dyspepsia. The appetite is usually diminished, and there is often a sense of pressure in the epigastrium after meals. There may be severe cardialgia. It is not always easy to determine its cause; often it seems to be purely nervous. Sometimes there may be myalgia. Moreover, chlorosis may be associated with a genuine ulcer of the stomach, as has been already mentioned. We believe, however, that this combination is much less frequent than is commonly supposed, for many physicians make a diagnosis of gastric ulcer upon insufficient data. The earlier assumption that the dyspeptic symptoms of chlorosis are associated with a lack of hydrochloric acid in the gastric juice is entirely incorrect. If we make a careful investigation of the gastric functions in chlorotic girls, we shall find that the amount of hydrochloric acid in the gastric juice is normal or even excessive. That gastroptosis may often be found in conjunction with chlorosis is not surprising (see Vol. I, page 565). An attempt has even been made to regard chlorosis as a result of gastroptosis, although, of course, this is entirely incorrect. The bowels are usually constipated in chlorosis, which is explained by the impaired appetite and the sluggish intestinal peristalsis. The spleen in chlorosis is rarely swollen to any considerable degree.

Over the cervical veins we often hear a loud murmur, which has been above mentioned as the *bruit de diable*. Examination of the heart often shows a slight increase in the area of cardiac dullness, although this does not seem due in most cases to an actual dilatation of the heart, but merely to a retraction of the borders of the lung. Anæmic cardiac murmurs are often heard, the pulse is accelerated and easily excited. Otherwise physical examination does not reveal any abnormal condition of the internal organs. It is a very great exception to find any indications of disturbance in the spleen or lymph-glands or the bone marrow. Fever is rare in mild cases of chlorosis, but in severe cases we sometimes see, especially at night, a slight rise of temperature, up to 101° F. (38.5° C.). The urine is usually pale, but seldom differs greatly, either in amount or constituents, from the normal character. It is noteworthy that chlorotic girls are very apt to suffer from disorders of menstruation. The menses may not appear until late, or they are very scanty. Even complete and long-continued amenorrhœa is often seen in chlorosis. All these disturbances seem to point to an essential relation between the appearance of chlorosis and the phenomena of sexual life. It is only in exceptional instances that chlorotic patients have menorrhagia.

Examinations of the blood have been made, in the hope of gaining a clearer insight into the true character of chlorosis.

Condition of the Blood in Chlorosis.—The blood is usually pale. Upon microscopic examination, we notice that the red blood corpuscles do not tend to form rouleaux so much as normally, and sometimes we can perceive that the globules are comparatively light-colored, and are not all of the same size (anisocythemia). Microcytosis and poikilocystosis frequently occur, particularly in severe cases of chlorosis. Abnormally large red corpuscles also appear, but seem to be rather the result of swelling than genuine macrocytes. Nucleated erythrocytes (almost always normoblasts) are seen only in small numbers, if at all. The red blood-cell count and the hemoglobin estimation generally show a condition which is characteristic of chlorosis—viz., less diminution in the number of red blood corpuscles than in the amount of hemoglobin. Hence the color index of the blood is lowered in chlorosis. We may even find the erythrocytes approximately normal in number (about 4,000,000) and hemoglobin only from fifty to sixty per cent. In most instances we find 3,000,000 to 3,500,000 red blood cells. In severe cases they are materially less, while the percentage of hemoglobin is reduced in still greater proportion. Obviously, therefore, the hemoglobin in the red blood cells is abnormally low in chlorosis. In most instances the white cells show no special changes, but leucopenia is occasionally observed. The number of the blood plaques is generally considerably increased.

We have already intimated that there is great variety in the general course of chlorosis. Many cases which seem severe at first terminate in complete recovery by the end of four to six weeks, or within a few months. Other cases are much more obstinate, resist all modes of treatment, and undergo frequent relapses; so that with its manifold variations, improvements, and exacerbations, the disease may occupy one or two years or more. The prognosis may be regarded as favorable on the whole, but it should always be given with a certain degree of reserve. It is true that ordinary chlorosis never involves direct danger to life. We shall, however, find that, so far as symptoms go, there is no sharp dividing line between "simple chlorosis" and "pernicious anæmia," and in any individual case it may not at first be possible to decide which variety of anæmia is before us.

There are hardly any true complications of chlorosis. If other organic diseases, such as pulmonary tuberculosis or gastric ulcer, exist along with chlorosis, one should always consider that the anæmia may have developed secondarily, or that the diseases may be present from mere coincidence. All the varieties of nervousness and hysteria are frequently found in association with chlorosis, and yet it cannot be assumed in every case that there is an essential causative relation between them.

The only remaining point worth mentioning is the occasional tendency of the blood to coagulate. Thus, thrombosis of a femoral vein occurs, or thrombosis of the vessels of the retina, and, what is a matter of clinical importance, thrombosis of the cranial sinuses (*q. v.*).

Diagnosis.—The diagnosis of chlorosis is, in a certain sense, easy, for the characteristic symptoms, the pallor, and the ordinary phenomena consequent on constitutional anæmia, are always easy to appreciate; but, on the other hand, the diagnosis is not perfectly simple, for the anæmia must be actually a primary essential anæmia in order to justify the name chlorosis. We therefore have no right to declare the diagnosis of chlorosis until we have made a careful and thorough physical examination, and have found that no factors are present which might produce a secondary anæmia.

We should, above all, bear in mind the possibility of incipient tuberculosis, and examine the lungs, the expectoration, and the general condition of the patient, and also consider hereditary and other predisposing influences. We should bear in mind the possibility of some organic disease of the stomach, such as ulcer, catarrh, or dilatation, or chronic renal disease, or possibly constitutional syphilis, giving rise to the syphilitic chlorosis before mentioned. In many cases it is easy to exclude all these secondary forms of anæmia, but occasionally the task is a very difficult one. In making a diagnosis of chlorosis it is very important, also, to consider always the general conditions, particularly the rapid onset of the anæmia at the time of adolescence in young girls, and the greater or less disturbance of menstruation. The characteristic condition of the blood (*vide supra*) is also to be considered.

Treatment of Anæmia and Chlorosis.—The first indication in treating chlorosis, as well as every other form of anæmia, is to promote the regeneration of the blood. There are two ways in which we can endeavor to fulfill this indication—first, by general hygienic and dietetic measures, and, secondly, by employing certain internal remedies.

First among general measures comes a care for pure air, sunlight, and proper food. Many a pale city girl regains her ruddy cheeks after a few weeks spent either in the country, or in the mountains, or at the seashore.

The choice of a place must, of course, depend mainly upon the circumstances of the patient. In many instances, boarding in any suitable country place answers as well as a long and expensive journey. If the seashore be chosen, some bathing resort on the North Sea will usually be preferable. There are other places where the patient can combine the advantages of pure forest air and ferruginous mineral water; these will be mentioned below.

The diet of anæmic patients should be easily digestible and rich in albumen; carbohydrates and fat should be given in but limited amounts to patients in whom the *panniculus adiposus* is already well developed. Lean patients, on the other hand, require such ingredients in their diet, and should be recommended to take simple puddings, extract of malt, butter, and cod-liver oil. Milk, if well borne, is certainly not an unsuitable food for anæmic patients, but we do not ascribe any special value to the drinking of large amounts of milk as a cure for chlorosis. In the first place, milk contains an extremely small amount of iron, and, secondly, it is very apt to cause troublesome constipation. The enforced use of milk is a torture to many chlorotic girls, and I have frequently secured the gratitude of my patients by relieving them from the milk for which they had an antipathy. Formerly it was not uncommon to order "milk cures"—i. e., an almost exclusive milk diet—but we regard this as decidedly unsuitable. On the other hand, we would especially advise the use of the yolks of eggs, provided eggs are well borne by the patient, and are not the object of strong aversion. The yolk of egg is comparatively rich in hematogen, which is a nuclein-like compound of iron from which it is probable that hemoglobin is formed; emphasis should likewise be laid upon the use of an abundance of green vegetables, such as spinach, for these all contain a large amount of iron. Some authors ascribe an exaggerated value to alcoholic beverages. They may be allowed in moderate amount if the patient asks for them and finds the appetite improve under their use. The best to choose for an emaciated patient are porter and other varieties of beer rich in extractive principles, but wine is often ill borne by chlorotic girls, being apt to cause palpitation, etc.

Many physicians also insist upon "abundant exercise in the open air." This can, however, be carried too far. A chlorotic girl is often made to take long walks in spite of her own reluctance to do so, with the result of becoming more languid and exhausted than she was before. We even believe that a case of profound anæmia will be benefited by a certain degree of bodily rest. Thus any needless tax upon the muscles is avoided. The most brilliant results we have ever observed in the treatment of chlorosis have been within the hospital. The patients, who are usually factory operatives and servant girls, are often kept in bed for the first two or three weeks. We admit that pure country air may be very beneficial, but we would strongly advise moderation in bodily exercise. We consider, therefore, rest in the open air, with appropriate nourishment, as one of the best remedies for chlorosis. If the patient begins to feel more vigorous, she will of herself take more exercise.

Of internal remedies, the preparations of iron have long occupied the first place in the treatment of all varieties of anæmia. How these exert their influence is at present quite uncertain, for the body needs so small an amount of iron that the iron contained in ordinary food seems to be quite sufficient for the demands of the system. The iron preparations seem to exert a specific stimulus on the blood-forming function of the bone marrow. The therapeutic efficiency of iron, however, is prominent chiefly in chlorosis; in all other forms of anæmia the efficacy of preparations of iron is much more uncertain and doubtful.

The number of iron preparations that are recommended and are used is very large, and is continually being increased by the commercial manufacturers of drugs, although there is no actual need of such increase. Almost every physician has his favorite prescription, to which he ascribes the greatest efficiency. We ourselves employ most frequently in chlorosis the long and well-known Blaud's pills. We can assure the reader that they are almost always well borne, and often cause a surprisingly rapid and decided improvement in chlorosis. We usually prescribe them in the following form: Dried sulphate of iron, pure carbonate of potassium, each 150 to 225 gr. (gm. 10 to 15); tragacanth, q. s. This makes 100 pills, of which 2 to 4 are to be given three times a day after meals. Other ferruginous remedies which are likewise often useful are reduced iron, which is absolutely pure iron in a very finely divided powder, and is prescribed as such or in pills, from 1 to 3 gr. (gm. 0.05 or 0.2), to be given several times a day; two preparations, ferri carbonas saccharata (U. S. P.), and ferrum oxydatum saccharatum solubile (Ph. G.), are prescribed as loose powders, of which .5 to 1 teaspoonful may be given in water three times a day. The last named has a special advantage in that it does not blacken the teeth. Another good preparation of iron is the lactate, which may be given in powders and pills, in the dose of 2 to 5 gr. (gm. 0.1 to 0.3). The tinctures of iron are, in general, scarcely to be recommended, and are usually employed only for children. Besides these long-tried iron preparations, modern industry has thrown upon the market an almost countless number of organic iron preparations in manifold combination and composition, produced mostly from the blood. Many of these remedies (ferratin and ferratose, triferrin, hemalbumen, hematogen, sanguinal, hemol, hemogallol, and many others) have acquired a certain reputation in practice, and we would not altogether question their efficiency. But no proof has ever been given that they yield better therapeutic results than the older simple preparations. At best, their higher price, and the more elegant appearance of the packages in which they are put up, are likely to create a certain suggestive effect with people of lesser intelligence.

Exceptionally, iron is not well borne, and causes digestive disturbances or diarrhea. We must then try another preparation or diminish the dose. It is usual to forbid the ingestion of sour articles of diet while taking iron, but the injunction is due mainly to prejudice.

The use of mineral waters containing iron is quite common, although the amount of iron thus introduced into the system is so small that it is hard to understand their therapeutic value. Of those artificially produced, that which contains the pyrophosphate of iron is the best, and seldom irritates even a delicate stomach. The natural chalybeate waters are also bottled and sent to distant places. They are often more effective if drunk upon the spot, but merely because, in that case, the general hygienic surroundings of the patient are better than they would be at home. The best-known and most popular ferruginous springs in Germany and Switzerland are situated at Cudowa, Rippoldsau, Elster, Schwalbach, Pyrmont, Steben, Kohlgrub, Driburg, Liebenstein, and St. Moritz. "Steel baths" are also much employed, but the effect is due not to the iron contained in the water, but to the temperature of the water and the carbonic-acid gas it contains. Baths generally seem to be of decided service in chlorosis, and hence we often prescribe simple baths or salt baths, giving a bath of fifteen or twenty minutes' duration, at a temperature of 90° to 95° F., two or three times a week, adding 5 or 6 pounds of salt to the bath. We not infrequently obtain very beneficial results from sweating, and especially from incandescent electric-light baths, which seem to have a direct stimulating effect on the formation of blood.

Some other remedies besides iron are occasionally employed in chlorosis. Of these, the chief is arsenic. Its effects are often very beneficial, and it deserves a trial, especially in severe cases. It may be given alone or in combination with iron. We have repeatedly seen cases of chlorosis which showed no improvement after the use of iron, in which the combination of iron with small amounts of arsenic, $\frac{1}{30}$ to $\frac{1}{15}$ gr. (gm. 0.002 to 0.004), three times a day, had a strikingly beneficial effect. The iron preparations are also frequently combined with quinin and other bitters; also with aperients (pil. aloes et ferri). Scholz and Strübing recommend the administration of sulphur in chlorosis—purified sulphur, 10 parts; sugar of milk, 20 parts; dose, as much as can be lifted on the point of a knife, three times a day. Other physicians extol manganese. No especial advantage, however, has been shown to pertain to these remedies.

Although the constitutional treatment is always the main consideration, yet sometimes special symptoms demand attention. Violent headache may be relieved by antipyrin, or similar remedies; dyspeptic symptoms are to be treated by careful injunctions as to diet. The patient should avoid pressure from without upon the region of the stomach, and should have easily digested food. The internal administration of hydrochloric acid is proper only when the examination of the gastric juice has shown that there is a diminution in hydrochloric acid; as a rule, there is an abundance of hydrochloric acid secreted (*vide supra*), and, if so, one might better try alkalies.

If there be constipation, it is desirable to remedy it rather by suitable diet than by laxatives. Thus, we may prescribe fruit, Graham bread, and other foods which promote peristaltic action by the mechanical irritation which they exert; also gentle massage of the abdomen, and only when these fail, injections or mild laxatives.

CHAPTER III

PROGRESSIVE PERNICIOUS ANÆMIA

(Grave Form of Essential Anæmia)

Definition and Ætiology.—We apply the term pernicious anæmia to a severe and rare form of anæmia which is usually fatal, and which develops as a primary disease, without demonstrable cause, in both men and women. We must hold fast to this conception of the disease, especially as the attempt has been repeatedly made of late to degrade pernicious anæmia from the rank of an idiopathic disease, and regard it as merely a grave form of secondary anæmia which may result from any one of the most various causes.

Of course, the diagnosis of primary anæmia may be wrongly made in cases where a more careful search will detect some special cause for the condition. Such a mistake may become evident at the autopsy. Thus, in repeated instances, cases which have been during life regarded as "pernicious anæmia" have been shown to be cancer of the stomach (Vol. I, page 548). In other cases, however, it is more difficult to discover the primary cause of the anæmia. Thus the profound anæmia which attacked the men at work upon the St. Gothard Tunnel was at first regarded as idiopathic, but more careful investigation showed that the disease was really due to anchylostomiasis (Vol. I, page 638), the ravages of which parasite have in other cases also been confounded with the symptoms of pernicious anæmia. Reyher and Runeberg have lately called attention to the fact that the presence of bothriocephalus latus (*vide* Vol. I, page 630) in the intestine may produce an affection resembling severe and apparently primary anæmia. Of late there have been a number of cases reported where the post-mortem examination showed that the progressive marasmus and anæmia of the patient had resulted from an extensive atrophy of the coats of the stomach and—what is more important—of the intestine, associated in some instances with well-marked lesions of the sympathetic plexus. These cases are very interesting, but have nothing whatever to do with pernicious anæmia. Often the symptoms even during life differ not a little from those of pernicious anæmia, so that we cannot think it right to establish what is called a "gastrointestinal variety of pernicious anæmia." It is a very interesting but somewhat puzzling fact that the disease may appear as a sequel to a great loss of blood, whether from one or several hemorrhages. It seems as if the body were sometimes unable to recover from the effects of large hemorrhages of this sort, so that the acute merges into a chronic and progressive anæmia, which, despite nursing and treatment, advances to a fatal termination. It is quite doubtful, however, whether such cases should be classed as genuine pernicious anæmia.

We, therefore, distinguish various kinds of secondary severe (pernicious) anæmias in which the anæmia is to be taken as a symptom or as a sequel to a primary disease of a different character (cancer, helminthiasis). In contradistinction to these, we designate as genuine pernicious anæmia that form of disease in which severe anæmia appears without any assignable cause and without any other primary organic affection, and in most cases advances steadily to death. The appearance of the blood and the general symptoms in the severe secondary anæmias correspond altogether with those of progressive pernicious anæmia. In spite of all this, it is absolutely necessary that these two conditions be entirely dissociated so long as we maintain the existence of a specific, progressive pernicious anæmia, the exact nature of which, it is true, is still unknown.

The credit of having been the first to study pernicious anæmia as a special form of disease belongs to Biermer (1868), although occasional instances of the disease had long before been noticed. [The puerperal form of pernicious anæmia was described by Walter Channing in 1842.]

With regard to the special ætiology of progressive pernicious anæmia, it must be confessed that we have as little definite information as about the ætiology of chlorosis. Often it has been thought that the disease might be infectious, but the discoveries thus far reported of specific microörganisms in the blood lack confirmation. We cannot at all find that the general course of pernicious anæmia points convincingly to the action of an exogenous, infectious, or toxic cause. The assumption is possible, but not unavoidable. Perhaps we have to deal with a purely endogenous disturbance of the blood life, or with anomalies of the internal secretion or internal cell life somewhat like the continued muscle waste that occurs in progressive muscular atrophy. There do not usually seem to be any special exciting causes. It has been maintained that unfavorable hygienic surroundings and insufficient nourishment promote the development of the disease; and this would apply perhaps to some cases, but certainly it does not to all.

In some cases the previous occupation of the patient appears to have had an injurious effect on the blood (toxic influences, such as carbonic oxid, also continued exposure to heat while working near fires, etc.). We are inclined to regard it as characteristic of this disease, as well as of chlorosis, that the anæmia may develop despite the most favorable outward circumstances. That pregnancy really produces a particular predisposition to pernicious anæmia, as Gusserow has maintained, we would not regard as certain. Among the many cases of pernicious anæmia which we have observed, there was not a single one arising from pregnancy. It is, of course, admitted that peculiar conditions of severe puerperal anæmia, with all the indications of pernicious anæmia, sometimes occur. They may end fatally, but may also undoubtedly entirely recover. There is, probably, some connection between them and specific toxic influences (analogous to the nephritis of pregnancy), and they thus present a special form of anæmia.

Genuine pernicious anæmia develops in males fully as often as in females. Most cases occur in middle life, from about the twenty-fifth to the fortieth year.

Pathology.—The principal criterion of progressive pernicious anæmia is a negative one—i. e., an entire absence of any organic disease (cancer, etc.), or of any condition (parasites) to which the origin of the anæmia might be ascribed. An absolutely positive diagnosis of "essential" anæmia can be made, therefore, only from the result of the autopsy. The positive anatomical conditions found have nothing peculiar to themselves; they are present in like manner in every case of severe ("pernicious") anæmia. We shall not here discuss the changes in the blood itself, inasmuch as they will more properly be described among the clinical phenomena. The changes in the internal organs appear, first of all, of course, as an excessive anæmia, and next as a fatty degeneration. The latter is usually best marked in the heart, but it also affects the kidneys, the liver, and the walls of the stomach and intestines, as well as the intima of the blood vessels. We have already pointed out that this fatty change is to be regarded as the direct result of the anæmia, probably because of the diminished supply of oxygen in the tissues.

The second important lesion of pernicious anæmia is the hemorrhages, usually small, but occasionally large; these are usually numerous, and are found in all sorts of locations. The most important of these hemorrhages are into the retina, because they can be demonstrated by means of the ophthalmoscope during life. They are very frequent. We may also find minute hemorrhages in the serous membranes, such as the pleura or pericardium, in the brain and in the mucous membranes. Cutaneous ecchymoses are also occasionally found. We shall revert later to the ætiology of these hemorrhages.

Still another secondary symptom is an abundant deposit of iron (hemosiderosis) in the cells of many organs. This can usually be determined only by the aid of the microscope or microchemical tests. It is most abundant in the peripheral zone of the hepatic lobules, but it also occurs in the kidneys, pancreas, bone marrow, spleen, stomach, and other organs. Quincke has studied this point very carefully, and finds that the total amount of iron contained in the liver in pernicious anæmia is much larger than normal. The most natural and most probable interpretation of this fact is that the iron originates from the destruction of large numbers of red blood corpuscles.

Besides the ordinary systemic conditions attendant upon the anæmia, special attention has naturally been directed to those organs which bear a close relation to the production of blood, but without finding any specific changes in them. The lymph-glands do not usually present any change in pernicious anæmia. The spleen is in many cases normal. In exceptional instances, however, it is decidedly enlarged, although never enormous. Even when the organ is enlarged there are no important histological changes to be detected. Cases of pernicious anæmia with marked splenic tumor are often termed splenic anæmia (*vide infra*), but we do not ourselves see how such cases differ essentially from others in which the spleen is not enlarged. The changes in the bone marrow are by far the most constant. This structure plays an important part in the formation of the blood. C. Wood, and after him Cohnheim, called attention to the fact that the bone marrow is almost invariably affected in pernicious anæmia. Instead of its normal yellow color, it has a dark-red color, like raspberry jelly. The normal "fatty marrow" of the long bones is transformed into red "lymphoidal" (i. e., blood-forming, the badly chosen name has nothing to do with the lymph!) marrow. This sign of regenerative activity is missing only in exceptional cases. Such cases have been termed the "aplastic" form of pernicious anæmia. In most instances we, however, find on microscopical examination of the red marrow every indication of an extremely active regeneration of the red blood corpuscles (numerous erythroblasts, nucleated normoblasts, megaloblasts, etc.). Obviously, however, this regeneration is only the reaction from an increased primary decay of the finished elements. Histologic conditions as well as the abundant deposits of iron in the bone marrow argue in favor of this conclusion. It is an interesting fact that signs of renewed blood-forming activity are also present in the other organs (liver, spleen, lymph-glands); thus we have a return to embryonic conditions (Erich Meyer and A. Heineke and others). The myeloid tissues of the bone marrow undoubtedly participate in these occurrences. Strange to say, the destruction of blood takes place chiefly in the blood-forming organs (bone marrow), and not in the blood itself, as in some toxic-hemolytic processes of a different character which lead to hemoglobinæmia (*q. v.*).

Symptoms.—As already stated, the symptoms of pernicious anæmia usually begin, independently of any demonstrable cause, in individuals previously healthy, and develop so slowly and gradually that it is hardly ever possible to determine the precise date of the commencement of the disease. This is still more likely to be the case if the trouble occurs, as indeed it may, in individuals who were previously feeble and pale without being actually ill. Occasionally a more acute onset is observed in pregnant women.

The first symptoms are almost invariably traceable to the incipient anæmia. They include the subjective disturbances and the objective changes which are seen in any ordinary anæmia. The patient feels languid and is easily fatigued; is liable to headache, vertigo, palpitation, and tinnitus aurium; there are anorexia, frequent nausea, and, above all, a striking pallor of the skin and mucous membranes, but while these symptoms in ordinary anæmia and chlorosis are moderate, in the severe forms of essential anæmia they are extreme.

In every well-marked case of pernicious anæmia the weakness of the patient is so great that he is permanently confined to bed, and standing or sitting for any length of time is possible. Usually the patient lies upon his back, with his head rather low, and presents a countenance of waxy pallor. Very frequently a slight but distinct yellowish hue may be detected. This yellowish hue may become so deep that, at first, the patients appear to be jaundiced. The urine, however, contains no bile pigment, and not much urobilin except in a fraction of the cases. Occasionally there are cutaneous ecchymoses, but these are exceptional. The mucous membrane of the lips, the gums, and the conjunctiva are likewise pale and colorless. The intellect is unimpaired, but all answers to questions are slow, apathetic, and delivered in a low and feeble tone of voice. The patient is usually incapable of any great mental exertion. He is sleepy, and often yawns. If the body be moved, and especially if an upright position be substituted for a horizontal one, there is great liability to syncope. This may result from any slight physical exertion, and is often accompanied by a peculiar spasmodic rigidity of the body. The main subjective symptoms are weakness, and more especially intense headache; this often assumes a pulsating character, and is located mainly in the temples or forehead. There is also an annoying ringing in the ears, described as a singing, rushing, or roaring sound. There are certain other subjective sensations—namely, nausea, a sense of thoracic oppression, and pain in the bones, which will be considered later on.

If we now proceed to a systematic physical examination, we are struck, in the first place, by the condition of the eyes. The pupils are often somewhat enlarged, but react in a normal manner. Vision is often disturbed by spots before the eyes. The anæmic amaurosis seen after a sudden large hemorrhage has not yet been observed in pernicious anæmia. The ophthalmoscopic examination of the fundus has very great diagnostic importance. We find in a majority of cases, although not in all, retinal hemorrhage. There may be either one or many hemorrhages. If they are extensive enough to involve the macula lutea or the disk, they may disturb vision greatly. Retinal hemorrhages invariably signify that the anæmia is profound, and are, with considerable justice, regarded as distinguishing pernicious from simple anæmia. Optic neuritis appears in some cases.

Respiratory Symptoms.—Breathing is usually accelerated, and, in the most advanced cases, is often remarkably deep and noisy (anæmic dyspnœa, *vide supra*). Sometimes there is a very annoying and almost painful sense of thoracic oppression, which is evidently connected with the dyspnœa. There is a "hunger for air." Physical examination of the lungs gives negative results. Sometimes there is a little cough, and there may be sufficient hemorrhage into the mucous membrane of the air-passages to give rise to a slight hemoptysis. Even in this case no anatomical changes can be detected during life. We may also mention in this connection that epistaxis is not very infrequent.

Phenomena referable to the *circulatory system* are of still greater clinical importance. The area of cardiac dullness is usually normal, although sometimes slightly increased. Upon palpation, we often find that the heart's action is exaggerated, and that its beat is felt over a larger area than normal. The pulse is usually decidedly rapid (100 to 120), but it is regular, and sometimes, but by no means invariably, small. It is often surprisingly strong. The loud "anæmic murmurs" are very characteristic. They can be appreciated at the apex of the heart, but are usually heard still better at its base. We generally hear a loud *bruit de diable* in the veins of the neck.

Digestive Organs.—The tongue is usually pale, smooth, and dry. The appetite is often very poor. The most prominent disturbances, however, are not due directly to the condition of the stomach, but they result from the cerebral anæmia—that is, they are symptoms of the irritation of the nervous centers. We refer to eructations and vomiting, which may be very frequent and distressing. There is generally a tendency to constipation. Occasionally there is diarrhea.

The *liver* is usually normal; as is also the *spleen* in many instances, while in others it is shown by percussion and palpation to be enlarged. It is sometimes possible, as we can ourselves bear witness, to observe an enlargement of the spleen increasing as the anæmia grows more profound, and again decreasing if improvement occurs (*vide infra*). On ordinary examination, the *urine* does not usually differ essentially from normal. With few exceptions it is free from albumen, and it never contains sugar. As already set forth, however, accurate quantitative analysis often furnishes important evidence of changes in tissue metamorphosis resultant upon the anæmia. We will merely mention once more the comparative increase in the amount of urea excreted, and the occasional excess of uric acid. Sometimes the urine gives a vivid reaction for indican.

In regard to what has already been said about the *marrow of the bones*, it is an interesting fact that they are very sensitive to pressure in many cases of pernicious anæmia. The sternum in particular is painful upon light percussion; and sometimes pressure will cause pain in the bones of the extremities. In rare instances swelling of the knee and other joints has been observed. A series of very interesting observations have been recently made by Lichtheim, Minnich, and others, which show that persons with primary or even secondary pernicious anæmia (e. g., anæmia due to the bothriocephalus) may quite suddenly develop well-marked *nervous symptoms*. These consist chiefly in paræsthesia in the hands and legs, in slight disturbances of sensation, unsteadiness and ataxia of the legs, weakness or complete abolition of the tendon reflexes, etc.—in brief, a group of symptoms resembling tabes. The pupils have been observed to become irresponsive to light. Upon the post-mortem examination of such cases, there is found, particularly in the posterior columns of the spinal cord, a well-marked disease, which consists for the most part in small scattered foci of degeneration, and in a more systemic (secondary) degeneration of the columns of Goll. Sometimes small capillary hemorrhages are also found. Slight changes of this sort may be observed in the spinal column, even when no special spinal symptoms had been present. Probably these conditions are due to toxic action.

The blood has been made the subject of numerous and careful investigations; nevertheless, no characteristic change has been discovered. The changes presented in progressive pernicious anæmia likewise occur in cases of profound secondary anæmia. The blood picture only proves that we have to do with a severe injury to the bone marrow, the cause for which can only be disclosed by the result of the entire clinical examination. To the naked eye the blood seems extremely pale and watery. The number of red blood corpuscles is sometimes so diminished that it seems incredible that life should persist. It is not at all unusual, in the worst stage of the disease, to find only 500,000 to 1,000,000 red blood corpuscles per c.mm., or even 250,000—that is, even less than one tenth of the normal amount. The proportion of hemoglobin in the blood is consequently much diminished to thirty to twenty per cent of the normal amount and even less.

Comparative determinations of the number of blood corpuscles and the amount of hemoglobin seem to indicate, however, that the individual blood corpuscles have not lost any of their coloring matter, and even present a particularly large amount of hemoglobin, a condition to which a compensatory significance is undoubtedly to be ascribed. The red blood corpuscles are found to present striking varieties in size and form (*vide* Fig. 108). While some corpuscles have a normal appearance, others may be of an unusually large size (macrocytes, megalocytes). These "giant corpuscles" appear normal except in dimensions, and some observers (Laache) have thought that they were even possessed of an unusual amount of hemoglobin (*vide supra*). In contrast with these large cells are found a varying number of minute red cells of a spherical shape: the so-called microcytes. Finally, there are numerous red blood corpuscles of abnormal shape. These Quincke was the first to notice. They present remarkable forms, being biscuit-shaped, hammer-shaped, or anvil-shaped, and so on, as illustrated in the accompanying cut. These "poikilocytes" are found in perfectly fresh undiluted blood, so that there is no reason to suppose that they are artificial products. The use of a basic stain (methylene blue) shows fine dot-shaped granules (so-called basophile granular degeneration) in some of the blood corpuscles.

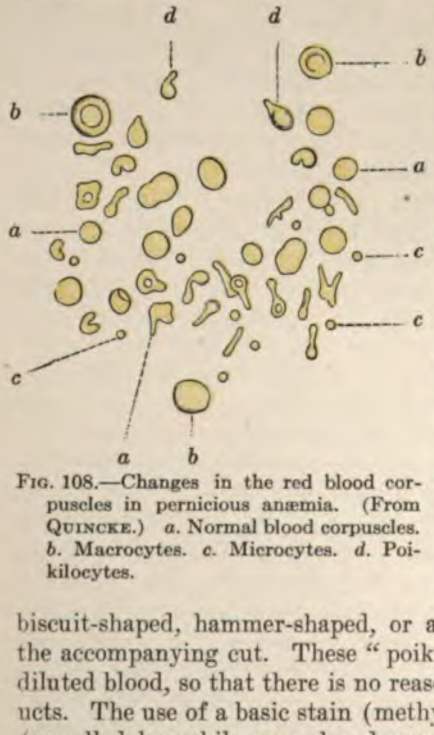

FIG. 108.—Changes in the red blood corpuscles in pernicious anæmia. (From QUINCKE.) *a*. Normal blood corpuscles. *b*. Macrocytes. *c*. Microcytes. *d*. Poikilocytes.

Besides these products of degeneration, we also find in the blood indications of an increased formation of new elements. We refer chiefly to the nucleated blood corpuscles. The appearance of megaloblasts in the blood, which Ehrlich explains as a reversion of the blood life to the embryonic state, has since his investigations been accepted as particularly characteristic of pernicious anæmia; but, according to my experience, they do not invariably appear. A similar explanation is given to the not infrequent occurrence of numerous polychromatophilic cells. At all events, we thus see the copious passage of unfinished elements from the bone marrow into the blood—in a manner, just as in war, the young and still untrained reserve troops must be sent to the front after heavy losses suffered by the army in the field. Only in cases of so-called aplastic pernicious anæmia (*vide supra*) are the regenerative elements entirely lacking in the blood. The white blood corpuscles are not usually increased in number; the number of polynuclear leucocytes in the blood is almost always even remarkably small. The number of lymphocytes, on the other hand, remains unchanged. Only in occasional instances has a leucocytosis been found, and then toward the end of life. Leube has described as leukæmia cases of pernicious anæmia with an increase of myelocytes in the blood. The number of blood plates is usually decidedly large. Chemical examination of the blood has not as yet brought to light any facts of great importance. It has been already stated that there is a great diminution in the total amount of hemoglobin. The amount of albumen in the blood serum remains nearly normal.

Finally, in discussing the constitutional manifestations of pernicious anæmia, mention must also be made of the behavior of the body temperature. As in all varieties of profound anæmia, the tendency to fever is very pronounced. In many cases the evening temperature will for weeks reach 100° to 101° F. (38° to 38.5° C.), but, occasionally, it may be even higher, reaching 102° F. (39° C.) or more. Previous to death, however, the temperature may become subnormal, falling to 86° F. (30° C.), or even lower.

General Course, Duration, and Prognosis.—As the very name "pernicious" indicates, the disease generally terminates unfavorably. Death usually seems to be the direct result of the extreme anæmia; special complications are exceptional. The disease often maintains a slow but gradual progress to the end. Its duration, reckoning from the appearance of the first symptoms, may not exceed three to six months. It may even run its course in a still shorter time. It seldom lasts more than a year. Sometimes its course is interrupted; there may be an arrest of the process, or improvement, or even apparent recovery. Usually, however, there are fresh relapses. In a certain class of cases the disease lasts two or three years, and is marked by a number of "attacks of anæmia" so intense that the subsequent improvement of the patient seems simply marvelous. It is in cases of this sort that splenic tumor has been made out at the time the anæmia was at its height; yet we do not perceive the necessity of establishing "splenic anæmia" as an affection essentially different from progressive pernicious anæmia. It is merely a clinical variety of the disease under discussion. Apparently, it likewise has an invariably fatal termination.

Permanent recovery may occur in cases of idiopathic anæmia so profound that we are at first inclined to regard them as pernicious. These cases are unfortunately very rare, and even when there is a marked improvement the danger of a relapse is to be borne in mind. The prognosis is therefore always very grave, if not absolutely unfavorable. Of course, general hygienic surroundings and good care may exert some influence upon the course of the disease. It is noteworthy that if pregnancy be complicated by profound anæmia there is a great liability to premature delivery, after which there is often a rapid change for the worse. There are exceptions to this rule.

Diagnosis.—It is seldom difficult to make out the existence of a profound anæmia, or to determine the degree of danger which the consequent symptoms indicate. We have, however, the same difficulty here as in chlorosis in proving that the anæmia is primary and idiopathic. The factors essential to this diagnosis have been already indicated. We should bear in mind the possibility of insidious tuberculosis, internal concealed hemorrhages, organic diseases of the stomach, septic subacute and chronic febrile endocarditis, certain cases of hepatic cirrhosis and certain parasites (anchylostomum, bothriocephalus), as the diseases which must be very carefully excluded in the diagnosis. From a practical standpoint, the question which comes most frequently into consideration is whether the condition is a pernicious anæmia or a latent gastric carcinoma. We cannot agree with certain enthusiastic hematologists that this differentiation can be made with absolute certainty in all cases by a consideration of the blood picture alone. It is certain, however, that besides all other clinical data, an accurate blood examination is of very great importance. Briefly recapitulating, the evidence in favor of pernicious anæmia is a relatively high color index, the presence of megalocytes; furthermore, and above all, of megaloblasts, and a diminished polynuclear leucocyte count. In the anæmia of carcinoma, all these peculiarities are, as a rule, lacking.

Treatment.—For treating progressive pernicious anæmia we have only the same remedies as for the benign variety of anæmia. We must provide the patient with nourishing and digestible food, and regulate all other hygienic matters; and in the way of medicine the greatest prospect of benefit lies in arsenic, while the efficiency of ferruginous preparations in pernicious anæmia is dubious. We may, however, try a combination of iron with arsenic. The best form in which to administer the arsenic is pills of arsenious acid (see prescriptions in the Appendix) or Fowler's solution. Subcutaneous injections of arsenic are also used a great deal, particularly injections of cacodylate of soda (cacodylate of soda, gr. xxxv to gr. lxxv [gm. 2 to 5]; carbolized spirit, 10 drops, aqua destill., ℥iijss. [gm. 100]; total daily doses of ⅓ to 1½ minims [0.02 to 0.1 c.c.], gradually increasing), or atoxyl (arsenic-acid anilid) in the same dosage. Besides arsenic, some physicians recommend phosphorus. We have seen in some cases considerable benefit from this (phosphorus, gr. 1/200 [gm. 0.0003] in keratin capsules, several times a day). The organo-therapeutic attempts with the internal administration of fresh bone marrow have as yet not given any satisfactory results.

If the case be not too far advanced, baths may prove useful adjuvants to the internal treatment. Salt baths or artificial carbonic-acid baths may be employed. Symptomatic treatment is often indicated: the dyspepsia may call for dilute hydrochloric acid, or the troublesome vomiting may require bits of ice, bromid of potassium, or opium.

The transfusion of blood has been employed in pernicious anæmia. Sometimes the effect seems to be favorable. Experience thus far, however, would not lead one to expect very great benefit from it. Von Ziemssen recommended in all severe forms of anæmia the subcutaneous injection of blood. It is done by injecting under the skin, by thoroughly disinfected instruments, 50 c.c.

of defibrinated human blood, divided into two parts, 25 c.c. being injected into each thigh. The point of injection is vigorously rubbed, and thus the injected blood is forced into the lymph channels. In severe cases this procedure should be frequently repeated. The results in several cases are said to have been very satisfactory, but, for reasons easily understood, the method has not found extensive practical application.

As an appendix, we desire briefly to mention the fact that, in children, not very infrequently severe anæmic conditions develop which are sometimes associated with hepatic and splenic enlargement, and which give various blood pictures. The accurate classification of these anæmias still offers great difficulties. A particular form of these anæmias of children has been described by von Jaksch under the name of infantile pseudoleukæmic anæmia. It is characterized by the occurrence of an unusually large number of nucleated red blood corpuscles (both normoblasts and megaloblasts). Associated with this is usually a distinct leucocytosis, and sometimes also a lymphocytosis. Spleen and liver are enlarged. The disease may last for years. A decided improvement may sometimes, but not always, be brought about by the administration of arsenic.

CHAPTER IV

THE LEUKÆMIC DISEASES

(Myelogenous and Lymphatic Leukæmia)

While in essential pernicious anæmia, in the main, only the erythropoietic portion of the bone marrow is diseased, the name leukæmia is applied to those morbid conditions in which the leucopoietic part of the blood-forming tissues is affected. Corresponding to these two entirely separate tissue systems, the myelogenous and the lymphatic, we also differentiate two forms of leukæmia, the myelogenóus and the lymphatic (lymphemia), each of which requires separate consideration.

1. MYELOGENOUS LEUKÆMIA (*Myelæmia*)

Conception and Ætiology.—The nature of myelogenous leukæmia consists in a disease of the myelogenous tissue which hypertrophies, and thus innumerable newly formed myeloid cells enter the blood. As the bone marrow is the main site of the myelogenous tissue, the pathological processes occur mainly there. Furthermore, corresponding hyperplasia and hypertrophy of the myelogenous tissue takes place in numerous other organs. This is particularly true in the spleen and liver, which, as already mentioned (page 42), possess during embryonic life myelogenous tissue and corresponding leucopoietic functions. This hyperplasia leads to large tumors of the liver, and, above all, of the spleen. These enlargements appear very much more prominently in the clinical picture during the patient's lifetime than do the changes in the bone marrow. This is the reason why formerly the leukæmic enlargement of the spleen particularly was looked upon as the essential anatomical basis of the leukæmia, and that the cases of leukæmia, associated with a large splenic tumor, were designated as "splenic leukæmia," a term that has now been entirely dropped. The occurrence of myelogenous hyperplasia in the various internal organs (besides the spleen and liver, also the lymph-glands, and rarely also the kidneys, intestines, etc.) has often also been interpreted as a form of metastatic formation, it being assumed that displaced bone marrow cells started up a development in the other organs. To our mind, the hypothesis that we have to deal with an independent general hyperplasia seems far more probable. Leukæmia is, to a certain extent, a "systemic disease" of the myelogenous tissue, in which, as is often the case in morbid process, a reversion to embryonic conditions takes place. In view of this, the assumption is also probable that the disease is not one of an exogenous nature, caused perhaps by parasites, but of a purely endogenous character, and possibly traceable to embryonic deviations. A corresponding, but, to be sure, still entirely hypothetical, conception of the nature of leukæmic diseases has been expressed by K. Ziegler. By exposing the spleen of mice and rabbits to the action of the Röntgen ray, he succeeded in producing an experimental myelogenous leukæmia. Ziegler explains this process by assuming that the myelogenous tissue of the bone marrow is, to a certain extent, stimulated to reaction by the damage done to the lymphatic tissue of the spleen. It begins to hypertrophy, myeloid elements enter the blood and also lodge in the spleen, where they further multiply. On this basis, in human leukæmia also, we might trace the proliferation of the myelogenous tissue to a primary weakness or injury of the lymphatic apparatus.

As may be gathered from the above, we are at present unable to speak of a definite ætiology of leukæmia. In the majority of cases the disease develops without demonstrable cause. At most, traumatic influences occasionally appear to play a rôle. Acute diseases (typhoid, malaria, syphilis, influenza) seem sometimes to be the exciting cause of the disease. Most of the cases of leukæmia occur during middle life. Occasionally, however, cases have been observed also in children. There seems to be no difference in predisposition in the two sexes.

Symptomatology.—The symptoms of leukæmia develop, as a rule, very gradually. Usually the patient first notices an increasing languor and weakness, which are frequently, but not always, associated with a striking pallor. Not infrequently the signs of a hemorrhagic diathesis (epistaxis, hemorrhage from the gums, etc.) become evident early in the disease. In the later stages the hemorrhages may become quite serious. Hemorrhages into the brain may lead to hemiplegia, or may be a direct cause of death. Hemorrhages of the stomach, intestine, kidney, and into the skin and muscles, have also been observed. We once saw a hemorrhagic tonsillitis with a fatal outcome from œdema of the glottis.

The splenic enlargement is the most striking change, clinically, in the internal organs. In the beginning this may be of slight degree, but finally it may reach such enormous dimensions that the spleen may reach far over into the right half of the abdominal cavity. As a rule, the enlarged spleen is of firm consistence, and presents a sharp medial edge, usually with one or two characteristic notches. In the beginning, subjective symptoms, particularly pain in the spleen, are usually only slight. With larger spleens, however,

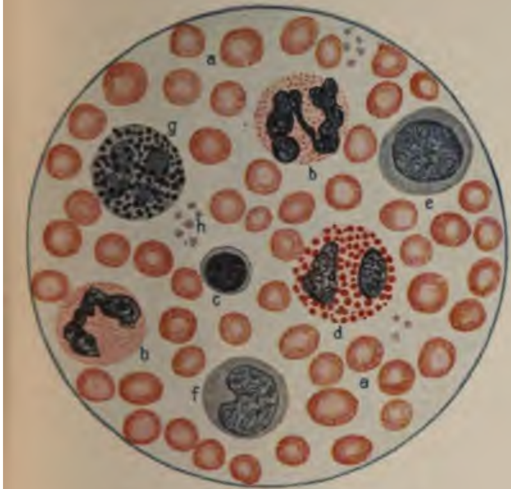

FIG. 1.

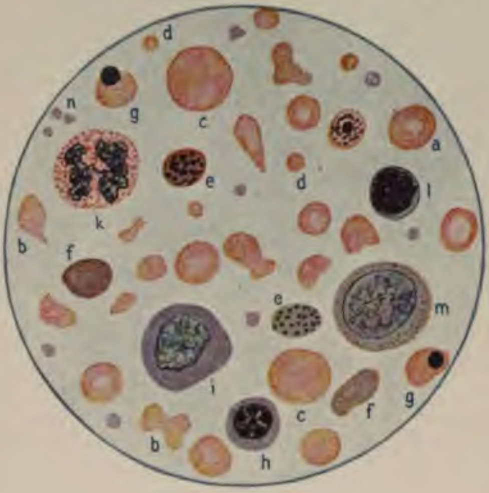

FIG. 2.

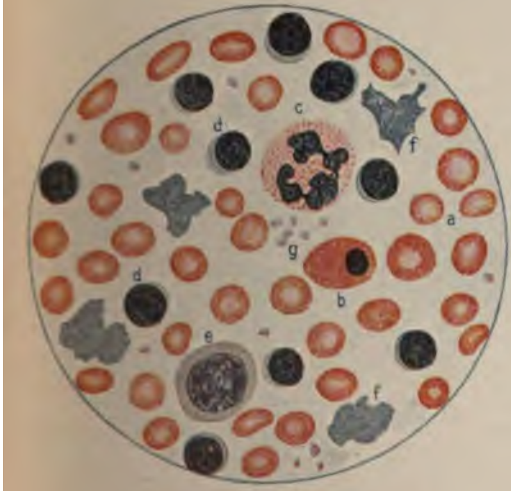

FIG. 3.

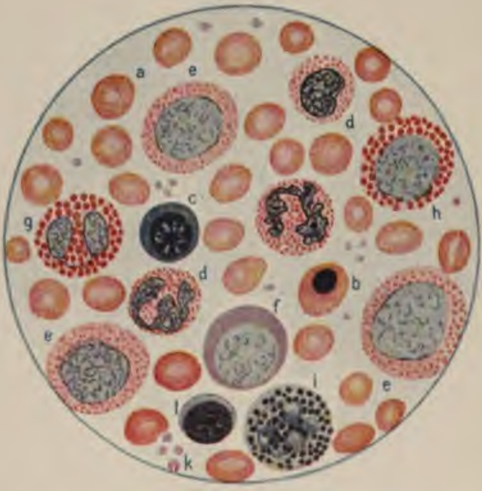

FIG. 4.

FIG. 1.—NORMAL BLOOD PICTURE (stained with eosin—methylene blue). *a.* Normocyte. *b.* Polymorphonuclear leucocyte. *c.* Lymphocyte. *d.* Eosinophile leucocyte. *e.* Large mononuclear, nongranular leucocyte. *f.* Transition form. *h.* Blood platelet.

FIG. 2.—PERNICIOUS ANÆMIA. *a.* Normocyte. *b.* Poikilocyte. *c.* Macrocyte. *d.* Microcyte. *e.* Stippled erythrocyte. *f.* Polychromatophilic erythrocyte. *g.* Normoblast. *h.* Erythroblast. *i.* Megaloblast (*h–i*, nucleated erythrocytes). *k.* Polymorphonuclear leucocyte. *l.* Lymphocyte. *m.* Large mononuclear, nongranular leucocyte. *n.* Blood platelet.

FIG. 3.—LYMPHATIC LEUKÆMIA. *a.* Normocyte. *b.* Normoblast. *c.* Polymorphonuclear leucocyte. *d.* Lymphocyte. *e.* Large mononuclear nongranular leucocyte. *f.* Lymphocyte shadow. *g.* Blood platelets.

FIG. 4.—MYELOGENOUS LEUKÆMIA. *a.* Normocyte. *b.* Normoblast. *c.* Erythroblast. *d.* Polymorphonuclear leucocyte. *e.* Myelocyte. *f.* Large mononuclear, nongranular leucocyte. *g.* Eosinophile leucocyte. *h.* Eosinophile myelocyte. *i.* Mast cell. *k.* Blood platelet. *l.* Lymphocyte.

there often occurs a very annoying, or even distressing, sensation of tension and fullness in the abdomen. Severe pains and signs of peritonitic irritation occasionally also occur as a result of the not uncommon occurrence of so-called splenic infarct—i. e., hemorrhage into the spleen and thrombosis of the splenic vessels. The respiration may be impeded by the upward displacement of the diaphragm. The liver, likewise, is usually felt to be enlarged, and sometimes so much so that its hard sharp edge may be felt rather low down in the abdomen. On the other hand, the lymph-glands are only rarely swollen.

The involvement of the bone marrow is usually noticed in the clinical picture only because of the changes in the blood (*vide infra*). As direct symptoms on the part of the bone marrow, the occasionally very severe bone pains might possibly be mentioned, and particularly the tenderness of the sternum to percussion. This symptom, however, may be entirely absent, even in severe myelæmia.

Of the remaining organic lesions the rather frequent retinal changes deserve mention. The background of the eye presents a "chocolate" color; the arteries and veins are differentiated with difficulty (so-called *fundus leucœmicus*). Besides this, we occasionally find grayish-white spots and streaks (*retinitis leucœmica*), and not rarely also retinal hemorrhages similar to those occurring in pernicious anæmia. Disturbances of hearing are also not uncommon (deafness, labyrinthine dizziness). Organic changes in the lungs are rare; hemorrhagic pleurisy is more common. Ascites, partly due to local congestion, and partly due to myelogenous changes in the omentum and peritoneum, is also occasionally observed. The urine is sometimes characterized by its relatively high percentage of urea—a sign of the increased destruction of albumen in the body. Probably associated with the excessive disintegration of leucocytes is the frequently very marked uric-acid excretion, indicated by a profuse urate sediment. The marked excretion of phosphoric acid perhaps depends upon the same cause.

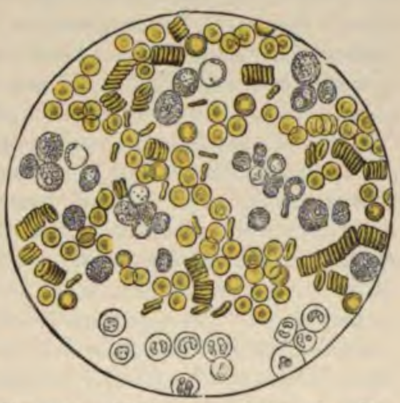

FIG. 109.—Myelogenous leukæmia. Unstained specimen. The nuclei of the leucocytes in the lower half of the specimen have been brought out by the addition of acetic acid.

From the diagnostic standpoint, the deciding feature is the very striking and interesting changes in the blood (Fig. 109 and Plate V, Fig. 4). Its color is often pale. In larger amounts of blood, for example in blood from the cadaver, a peculiar brownish-white or chocolate-like color of the blood is often noticeable. On more accurate microscopic examination, we can usually at the first glance detect a frequently enormous increase in the white blood cells, which may reach as high as 200,000 to 500,000 per c.mm. The deciding point, however, is not the number but the type of cells. The cells are almost exclusively of the myeloid variety, there being, first of all, numerous ordinary polynuclear neutrophilic leucocytes. Besides these, eosinophile cells are always present, and not infrequently an increased number of basophile leucocytes. Then we also find a large number of cell forms which, under normal conditions, exist only in the bone marrow itself, and do not enter the blood. There are, above all, very many myelocytes with various granulations, and also myeloblasts and other transitional forms ("large mononuclears," etc.), besides all kinds of abnormal cells. In contrast with these findings, which clearly point to a severe disease of the leucoplastic-myelogenous tissue, the involvement of the erythroplastic tissue stands decidedly in the background. The latter, however, especially in the later stages of the disease, even if perhaps in part as a secondary phenomenon, is often enough affected. The red blood-cell count is usually reduced to about 2,000,000 or 3,000,000. Anisocytosis and poikilocytosis in moderate degree are usually present. Isolated normoblasts are also rarely absent. The hemoglobin content of the blood shows, on the whole, a moderate reduction. Naturally, the lymphocytes appear relatively very much in the background of the blood picture. Their absolute number, however, is nearly normal, or even somewhat increased. The number of blood plaques is generally decidedly increased.

It is obvious that such a changed condition of the blood must have a decided effect upon the general condition of the patient. Still, it is worth noting that nothing is known of the effects of the myelæmia, as such, and that the constitutional symptoms in leukæmia seem to be dependent, in the main, upon the accompanying anæmia. With simultaneously severe anæmia, the constitutional manifestations of anæmia are prominent in the same manner in leukæmia as in all severe anæmias. They are evident in the visible pallor of the skin which in advanced cases may reach as high a grade as in the pernicious anæmias; furthermore, in the anæmic murmurs in the heart and the veins of the neck, the general weakness and fatigue, the anorexia and digestive disturbances, the palpitation and dyspnœa, and finally in the long list of anæmic brain symptoms—i. e., headache, dizziness, attacks of syncope, tinnitus, etc. Sometimes the patients suffer from pronounced itching of the skin. As long as the anæmia and its consequences do not become pronounced, the general condition of the leukæmic patient may be comparatively satisfactory. A symptom of leukæmia, repeatedly observed in the male, is priapism. This phenomenon may sometimes, as we have observed, be the first symptom of the disease noted by the patient. It is not due to a nervous irritation, but to the formation of white thrombi, rich in leucocytes, in the corpora cavernosa (Kast). In isolated cases multiple paralyses of the cranial nerves (facial, hypoglossal, auditory, etc.) have been observed. These depend partly upon small hemorrhages and partly upon degenerative changes in the medulla.

The temperature shows the same tendency to elevation as in the severe anæmias. In advanced cases of leukæmia we occasionally even observe very high intermittent elevations of temperature, up to 103° to 104° F. (39.5° to 40° C.). These are sometimes accompanied by pronounced chills, while the subsequent drop in the fever is associated with a severe and very exhausting perspiration. These high temperatures are probably due to toxic products of the decomposition of blood cells (so-called ferment intoxication). Complications, which are not directly connected with the leukæmia, are generally rare. Still, intercurrent acute diseases (pneumonia, pulmonary tuberculosis, etc.) are occasionally seen. It is very remarkable that the number of white blood cells sometimes returns entirely to the normal during such a complication.

The general course of myelogenous leukæmia is usually very chronic. The condition often lasts several years. Spontaneous and decided remissions, as in pernicious anæmia, scarcely occur. The final outcome is always unfavorable. Death is the result of the increasing general weakness, hemorrhages (*vide supra*), or secondary complications. Isolated cases run such a rapid fatal course (in a few weeks or months) that we may speak of an acute myelogenous leukæmia. The general clinical picture in many respects, then, resembles acute lymphatic leukæmia, which will be later described. The different blood picture and the absence of lymphatic enlargements differentiate it from the latter disease. The numerous large mononuclear transitional cells are characteristic of myelogenous leukæmia.

Pathological Anatomy.—There is little to be added regarding the pathological anatomical findings in myelogenous leukæmia, especially as we cannot here go into histological details. The chocolate-colored blood, the presence of many puriform clots, the large splenic tumor depending upon the enormous proliferation of the myelogenous tissue, the corresponding changes in the liver and other organs, have all been mentioned. The spleen may weigh from 6 to 11 pounds (3 to 5 kg.), and may be over 12 inches (30 cm.) long. Its cut surface is usually rather deep red, but also often of a lighter, more reddish-gray color. Not infrequently, large bloody infarcts in the form of circumscribed dark-red foci are found in it. The bone marrow shows a red or a grayish-red or yellowish (purulent) color, and on microscopic examination all forms and varieties of myeloid cells are found in the greatest profusion. Very frequently after death the so-called Charcot's crystals are found in the bone marrow and also in the blood—the same octahedral crystals that are also found in the expectoration in bronchial asthma. Their formation probably depends upon the disintegration of the eosinophile cells.

Treatment.—The general treatment is the same as that of severe anæmia, and therefore requires no detailed description here. Of internal remedies, the iron preparations have no effect, while arsenic is sometimes of undoubted value. It is prescribed in the manner described above (page 66), and at times a decided improvement in the general condition and the blood picture, as well as a diminution in size of the splenic tumor, are noticed. At present, however, by far the most important and interesting treatment in leukæmia is that by the Röntgen rays. This was first recommended on a purely empirical basis by American physicians (Senn and others), and then received theoretical support when H. Heineke proved that the X rays are able in a most elective manner to destroy certain types of cells. We consider it one of the most interesting therapeutic observations to see how a large leukæmic tumor of the spleen can be reduced to one half its original size, or even less, by regular X-ray exposures; how the leucocyte count will fall from 250,000 to 30,000 and less, even after a few weeks, and, what is most important from the practical standpoint, how the general condition and strength of the patient are at the same time decidedly improved thereby. It must be noted, to be sure, that the improvement is often only temporary, and that actual cures of leukæmia have not yet been accomplished. Nevertheless, we know of several instances in which severe cases were so much improved that the patients were again able to do rather heavy work for several years, notwithstanding the persistence of slight objective changes. At any rate, at least, an attempt with X-ray treatment is urgently indicated. The treatment must, however, be carried out by an experienced radiographer, and under the constant control of blood examinations. Usually the spleen and also the various long bones are alternately X-rayed every day or two for about ten to fifteen minutes or longer. After about twenty times, there should be a pause in the treatment, which is then to be resumed at definite intervals.

The extirpation of the splenic tumor should be absolutely discarded as most dangerous and entirely useless. Symptomatic improvement is said to have been frequently obtained from oxygen inhalations.

2. LYMPHATIC LEUKÆMIA (*Lymphæmia*)

Conception and Ætiology.—While myelogenous leukæmia is due to a disease of the myelogenous tissue, and is characterized by a resulting increase of the myeloid cells in the blood, lymphatic leukæmia is due to a hyperplasia of lymphatic tissue, and is characterized by an increase of the lymphoid cells (lymphocytes) in the blood. Hence the lymph-glands, above all, present a decided hyperplasia. In the neck, axilla, and groins, these increase in size and form large tumors. The same enlargement often also involves the internal lymph-glands, as well as the other lymphatic organs (tonsils, mucous-membrane follicles, etc.). The enlargement of the bronchial and retrobronchial lymph-glands can be definitely demonstrated by the abnormal dullness, but, above all, by X-ray examination. The spleen (the "lymph-gland of the blood") is also almost always distinctly enlarged, though such large splenic tumors as occur in myelogenous leukæmia are exceptional. Still, we saw a case of pronounced lymphatic leukæmia in which only a large splenic tumor, with practically no lymph-gland enlargement, was demonstrable. In almost all the other organs (liver, skin, bone marrow, etc.) there is also a lymphoid proliferation, so that, as a matter of fact, we may designate lymphatic leukæmia as a systemic disease of the entire lymphatic apparatus of the body.

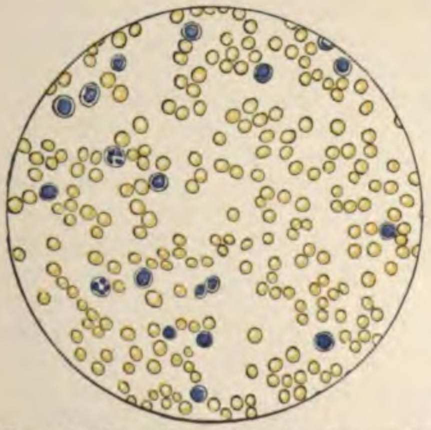

FIG. 110.—Lymphatic leukæmia. Increase in the lymphocytes. (After RIEDER.)

A causative factor for the development of the disease is almost never demonstrable. The malady occurs in children and in adults, and sometimes even in advanced age. Usually the striking enlargement of the external lymphatic glands is the symptom which brings the patient to the physician. Besides this, there are often distinct signs of anæmia and general weakness. The diagnosis can be made only by the microscopic examination of the blood. What is at once evident is the marked increase in the white cells, or, rather, in the lymphocytes (see Fig. 110 and Plate V, Fig. 3), the number of which may reach as high as 200,000 or more per c.mm. For the greater part these are the usual small lymphocytes, but very much larger ones, and other atypical cells, as well as so-called "lymphocyte shadows" (Plate V, Fig. 3 *f*), may be present. The ordinary polynuclears are not increased in number, as a rule. Abnormal myeloid cells are absent. The red blood cells may, for a long while, remain almost normal in count and quality. After the disease has continued for some time, a pronounced anæmia, with corresponding blood changes, usually develops.

Course of the Disease.—In the majority of cases the disease is a very chronic one. Besides the clinical symptoms already mentioned, we must still note the not uncommon changes in the skin (prurigo-like diseases, etc.), albuminuria, retinitis, accompanying ear affections, and, above all, the tendency to hemorrhages (epistaxis, etc.). The hemorrhagic diathesis is particularly pronounced in those cases which, on account of their rapid course, are designated as acute lymphatic leukæmia. These cases begin like an acute infectious disease with febrile constitutional manifestations, to which usually are added the signs of a hemorrhagic diathesis (bleeding from the nose and gums, hemorrhagic sore throat, retinal hemorrhage), and then multiple lymph-gland enlargements and splenic tumor. The face becomes dull and pale, there are severe headaches, and the temperature may rise irregularly as high as 104° F. (40° C.), or higher. The diagnosis is made only by the blood examination. In the beginning the blood shows a moderate lymphocytosis, which, however, rapidly increases. If the cells are typical small lymphocytes, the decision is not difficult. If, however, the blood contains in the main so-called large lymphocytes with atypical forms, it may be difficult to decide whether we are dealing with lymphoid or myeloid cells. In true lymphæmia the bone-marrow cells remain quite in the background. The condition of the erythrocytes often corresponds to the picture of a pernicious anæmia.

Prognosis and Treatment.—The prognosis of these acute cases is absolutely unfavorable. Death occurs in a few weeks with signs which resemble the severest septic infection, and which are really, in part, the result of a secondary sepsis. Attempts at treatment (arsenic) are almost always without result. The nature of these remarkable cases, and their relation to the other forms of leukæmia, are still entirely in the dark. In many respects they present quite the appearance of a severe acute infectious disease. A sharp limitation and differentiation of the cases brought under this category is still very uncertain.

The treatment of chronic lymphæmia is very much more promising than that of the acute variety. With arsenic and systematic X-ray treatment (*vide supra*) of the lymph-glands and splenic tumor, quite satisfactory improvement can be brought about, at least temporarily.

In close relation to acute lymphæmia is a peculiar, rare disease, *chloroma*, so called on account of the green color of the lymphatic tumors, which develop usually in a rather acute manner in the lymph-glands, spleen, liver, etc., but, above all, quite characteristically in or below the periosteum of the skull, spine, ribs, and other bones. The serious, often febrile general condition, the anæmia, the tendency toward hemorrhages, and, above all, the usually marked lymphæmic condition of the blood, indicate the close relationship of chloroma to lymphatic leukæmia.

CHAPTER V

THE PSEUDO-LEUKÆMIC DISEASES

(Lymphatic and Splenic Pseudo-leukæmia. Adenia. Lymphadenia. Malignant Lymphoma. Hodgkin's Disease. Banti's Disease)

Cohnheim has included under the caption of "Pseudo-leukæmia" a number of morbid conditions, in which apparently the same pathological changes occur in the organs as in the true leukæmias, while the blood usually shows a diminution in the number of red blood corpuscles, but no—or, at least, no decided—increase in the number of the white cells. As to the more exact nature of these diseases, little is known with certainty, and there can be no question that a number of entirely different morbid processes have been grouped together under the heading of "Pseudo-leukæmia." Only an accurate study of numerous individual cases will make it possible to bring order and system into this multiplicity of clinical pictures. For the time being we must be satisfied with describing the clinical syndromes as they occur in practice, according to their course and symptoms. It might also be advisable to mention the most important general view points, on the basis of which an attempt has been made to bring order into the confusion that has existed hitherto.

1. **Lymphatic Pseudo-leukæmia.**—This is by far the most frequent of the diseases which come under consideration here. It was first described by Hodgkin in England in 1832, and is therefore still frequently called Hodgkin's disease. Wunderlich was the first in Germany to study the disease thoroughly; he described it in 1858 under the name of "progressive multiple hypertrophy of the lymph-glands," and later Billroth termed it "multiple malignant lymphoma." Trousseau gave it the name of "adenia."

Lymphatic pseudo-leukæmia is characterized by the occurrence of multiple lymph-gland enlargements, combined with a gradually increasing general anæmia and cachexia, but without lymphæmic blood findings.

The disease usually occurs in youth and middle life. It is undoubtedly somewhat more common in the male than in the female sex. Particular ætiological factors for the disease can almost never be demonstrated. In individual cases the malady seems to develop secondarily to some other disease (suppurative otitis, acute infectious diseases). The first noticeable symptom is generally the occurrence of painless enlargements of the lymph-glands of the neck, angle of the jaw, back of the neck, etc. The glands enlarge slowly, and finally they may develop into large deforming tumors. In the meanwhile glandular tumors have also developed in other parts of the body (the axilla, inguinal regions, etc.). In many cases the internal lymph-glands and lymphatic apparatus are also diseased. According to our experience the tonsils are rarely involved, but, on the other hand, the mediastinal, bronchial, and retrobronchial lymph-glands are frequently implicated. Although until now the affection of these glands was only rarely recognized by percussion, or through the secondary symptoms of compression (*vide infra*), we have at present in the radiography of the thorax an extremely important and useful aid in the recognition of these conditions. The mesenteric and retroperitoneal glands also are not infrequently diseased, even if they can only exceptionally be demonstrated as palpable tumors. At autopsy most remarkable lymphatic neoplasms are occasionally found in the liver, intestine, kidneys, etc. In the majority of instances the spleen also is distinctly enlarged, although the splenic tumor never reaches such dimensions as in true leukæmia, or in so-called Banti's disease. As a rule, however, the spleen is distinctly felt to be moderately enlarged.

All these lymph-gland enlargements may be present a long time without producing decided symptoms. The pseudo-leukæmic lymphomata are never painful. On the other hand, under certain conditions pronounced pressure effects upon neighboring organs may develop. These are, especially, dyspnœa from compression of the trachea and of separate bronchi, dysphagia, œdema from pressure upon the veins, ascites, icterus, etc. With considerable enlargement of the intrathoracic lymph-glands, the subcutaneous veins of the thoracic wall usually become prominent. As the glandular enlargement increases, a pronounced general bodily weakness and anæmia develop. The question as to whether we are dealing with a lymphatic leukæmia or with a pseudo-leukæmia can naturally only be decided by an accurate blood examination. In pseudo-leukæmia there is often, but not always, found a more or less pronounced oligocythæmia (about 2,500,000 to 3,000,000 red-blood corpuscles to the c.mm.). With this there is no increase in the number of lymphocytes, the count of which is usually even strikingly low. There are no myelocytes, but not infrequently there is a simple leucocytosis, particularly during febrile periods, just as we find a leucocytosis occurring in other febrile conditions (*vide infra*).

Of the other symptoms, the behavior of the body temperature deserves special consideration. Not infrequently, periods of two or three weeks with temperature elevations of 102° to 104° F. (39° to 40° C.) alternate with intervals of almost normal temperature. Such observations were first described by Pel and Ebstein under the name of "chronic recurrent fever." As occasionally in these cases only the internal glands were swollen, the clinical picture could only be interpreted with difficulty during the lifetime of the patient. We, however, have also observed these same febrile attacks in the ordinary form of pseudo-leukæmia. Sometimes these elevations of temperature are associated with severe abdominal pain, which may be due to an acute increase of the glandular enlargement. Persistent remittent or intermittent fever may also occur. The pulse is usually rapid. Anæmic cardiac murmurs are occasionally heard. In some cases a hemorrhagic diathesis develops. We have noted with comparative frequency pruriginous dermatoses. These consist in the development of small, very itchy nodules (lymphomata) in the skin of the trunk and extremities. The urine usually shows no special changes. We, however, have once noted a persistent albuminuria.

Sometimes the disease runs a comparatively rapid course, lasting only a few months. In other cases it may last several years. Periods of more rapid progression of the disease alternate with others in which the malady seems to be at a standstill. Actual cures are rare (*vide infra*), so that in general an unfavorable prognosis must be given. Still, decided improvements may occur. Death results from the increasing general weakness and anæmia, the hemorrhages, compression symptoms (dyspnœa, hydrothorax), or from accidental complications.

Pathology (Kundrat, Sternberg, Ribbert, and others) has at least increased our knowledge of the pseudo-leukæmic diseases in so far as it has proved the necessity of the division of lymphatic pseudo-leukæmia into at least two provisional groups. In the first group of cases the pathological process in the lymph-glands consists exclusively in a true lymphocyte proliferation, while in the second group the lymph-glands show a growth of inflammatory granulation tissue in which all varieties of cell forms occur (round cells, polymorphonuclear leucocytes, giant cells, epithelioid cells, etc.). The "true" pseudo-leukæmias belong to the former group. In these cases there is a chronic disease of the entire lymphatic apparatus, in which, however, for some unknown reason no lymphæmia develops, although a relative lymphocytosis is often demonstrable. Besides this general lymphomatosis, a more local glandular disease also occurs, which can then be designated as lympho-sarcomatosis. To the second group of multiple glandular disease belong especially those cases associated with occasional febrile attacks in which there is a reduction in the number of lymphocytes, as an expression of a defective lymphatic apparatus, but at the same time there is not uncommonly a leucocytosis. The ætiological position of these cases is still in doubt, but the assumption of an infectious cause is probably not far from correct. Some observations speak decidedly in favor of a close relationship with tuberculosis (the demonstration of isolated tubercle bacilli, other tuberculous lesions) and perhaps sometimes also with syphilis.

In the treatment of lymphatic pseudo-leukæmia, there are two remedies at our disposal which have proved useful in many cases, viz., the preparations of arsenic and the Röntgen rays. The arsenic treatment is carried out in the same manner as described in the treatment of anæmia (*vide supra*, page 66). Arsenic is given internally in the form of Fowler's solution, or in pills, or it is given hypodermatically in the form of cacodyl or atoxyl. The Röntgen treatment consists in exposing the glandular tumors to the action of the X ray. An exposure of ten to fifteen minutes is given daily. After twenty to twenty-five exposures, the treatment is stopped for a time, during which interval arsenic may be given. The treatment must be frequently repeated. The results are not the same in all cases. Sometimes we see a most striking recession of the glandular enlargements, even including those of the bronchial glands, and at the same time improvement in the general condition. In other cases there is little effect. In general the results of arsenic and X-ray treatment seem to be decidedly better in the lymphomatous than in the granulomatous type of the disease. At any rate the X-ray treatment should be tried in every case. Technical details should be sought for in special works on the subject.

Besides this, great attention should naturally be given to the general treatment. Nutritious diet, a stay in the country, salt-water baths, carbonic-acid baths, and the like, are especially to be considered. External local applications have little effect. Still, inunctions of green soap, iothion salve, etc., may be tried.

The operative removal of the glandular tumors might possibly be tried in the very inception of a localized process. In all advanced cases surgical treatment is contraindicated, as it has been repeatedly observed that the condition has gotten worse after operative interference.

2. **Splenic Pseudo-leukæmia. Splenic Anæmia. Banti's Disease.**—At least a portion of the cases included under the heading of lymphatic pseudo-leukæmia represent a definite clinical entity, so that, as just noted, only in exceptional instances is the differentiation from the other forms of multiple lymphomatosis (tuberculosis, syphilis, lympho-sarcoma) difficult. On the other hand, an exact definition and differentiation of the cases belonging to the group of splenic pseudo-leukæmic diseases is, at the present time, utterly impossible. Neither from the clinical nor the pathological, to say nothing of the ætiological aspect, can definitely limited pictures of disease be constructed. For the time being we must be satisfied with the fact based upon experience that there exist peculiar chronic morbid conditions characterized by splenic enlargement and, frequently, simultaneous enlargement of the liver, in which the blood may, to be sure, show a variety of anæmic changes, but, at all events, no characteristics of leukæmia. Certain cases of "splenic pseudo-leukæmia" are found at autopsy to belong probably to the "lymphatic pseudo-leukæmia" type. The enlargement of the external lymphatic glands is, indeed, absent, but the mesenteric glands are enlarged, and the changes in the liver are quite the same as in the ordinary pseudo-leukæmia.

In conclusion we must refer to the so-called Banti's disease. Under the name of "splenomegaly with hepatic cirrhosis," Banti described a peculiar affection characterized by the development of a primary chronic splenic tumor, associated with a certain amount of cachexia and anæmia. Later on an enlargement of the liver develops, which gradually turns into hepatic cirrhosis with ascites. Banti assumed that the liver disease was the result of the action of toxic products formed in the diseased spleen, and carried to the liver by the portal vein. He based this hypothesis partly upon the fact that decided atheromatous changes have been found in the splenic vein, and partly on the interesting observation that in many of these cases extirpation of the spleen brought about a cure. These communications of Banti have certainly stimulated great interest in the subject, but the question is by no means settled. The talk about the cures after extirpation of the spleen has also subsided considerably.

APPENDIX

POLYCYTHÆMIA WITH SPLENOMEGALY

We must still mention a peculiar morbid condition, first described by French authors (Vaquez, Rendu, and Widal), but of which quite a number of cases have also already been observed in Germany (Osler, Türk, Weintraud, etc.). The patients are usually adult males, who complain of headache, congestion, dizziness, dyspnœa, vomiting, general weakness, and the like. On examination, the most striking feature is the cyanotic appearance of the patient, although there is no cardiac or pulmonary lesion. The examination of the blood shows an unusually great increase of the red blood corpuscles. Ten million or more erythrocytes to the cubic millimeter have been found. The hemoglobin content of the blood is absolutely high, but in relation to the blood count rather decreased. The leucocyte count is either nearly normal or somewhat increased. The spleen is usually felt distinctly enlarged. Albuminuria is sometimes present. The tendency to hemorrhages from the gums, nose, hemorrhoids, etc., is striking. The actual nature of this remarkable disease—to a certain extent a newer, more scientific formulation of the old conception of plethora—is not yet cleared up. Still, some findings speak for an abnormal increase in the hematopoietic function of the bone marrow—above all, the almost constant occurrence of nucleated red blood corpuscles in the blood, and also the not infrequent accompanying moderate leucocytosis, already referred to. From the therapeutic standpoint, occasional good results have been observed from frequently repeated small venesections.

CHAPTER VI

HEMOGLOBINÆMIA AND HEMOGLOBINURIA

Definition and General Ætiological Considerations.—If any cause produces a solution of the red blood corpuscles in the blood serum, hemoglobin is excreted through the kidneys. The hemoglobinæmia—the presence of free hemoglobin in solution in the blood—occasions hemoglobinuria—i. e., the excretion of hemoglobin in the urine. The causes of hemoglobinæmia and its correlative hemoglobinuria are manifold. In the first place, there is a whole series of poisons which, if introduced into the blood in sufficient amount, exercise a directly destructive influence upon the red blood corpuscles, and thus excite hemoglobinuria. To this list belong chlorate of potassium (Marchand), pyrogallic acid and naphthol (Neisser), sulphuric acid, glycerin, toluylendiamin, and many other substances. Distilled water is also in this sense a poison. Boström has discovered a fact of practical importance which deserves mention in this connection. It is that a certain kind of mushroom (*Helvella esculenta*), when fresh, contains a poison which is capable of producing intense hemoglobinuria, and such grave symptoms as jaundice, delirium, drowsiness, and tetanic convulsions, with perhaps a fatal termination. This poison is, however, so evanescent and so readily soluble in hot water that the mushroom becomes perfectly harmless if thoroughly soaked and then boiled, or if it has been dried.

Secondly, hemoglobinuria may be developed in connection with infectious diseases. In this case, also, it is probably referable to the action of poisons created within the system. Thus, hemoglobinuria has been observed in the course of a severe attack of scarlet fever or typhoid fever. Possibly malarial poisoning and syphilis may give rise to paroxysmal hemoglobinuria. This question will be discussed later on.

There is a third mode of origin which also possesses practical importance. If blood from one animal be injected into another of a different species, hemoglobinuria is almost sure to result. Not only do the injected blood corpuscles undergo solution, but also the injected serum acts as a poison upn the original blood corpuscles, destroying and dissolving them. This transfusion-hemoglobinuria has been described by Prévost, Dumas, Ponfick, and Landois. It can be realized in human beings, as there was only too good opportunity to observe during the brief period when the transfusion of lamb's blood was in vogue. The practical deduction is evident that we should not use for injection into the circulatory system of a patient anything but an unirritating salt solution or human blood. [Hemolysis has followed the transfusion of human blood. It is safer to establish the suitability of the donor by a preliminary test.]

A fourth and very important ætiological factor is exposure to extremes of temperature. Hemoglobinuria follows extensive burns. The blood corpuscles in that region of the periphery exposed to the heat are destroyed. Cold is capable of producing precisely analogous results. This is particularly evident in the cases of so-called paroxysmal hemoglobinuria described by Wickham Legg, Lichtheim, Murri, Küssner, and others. [Raynaud's disease (see page 565) and hemoglobinuria are associated in a sufficiently large proportion of cases to show some relationship between the two.]

Pathology and Symptoms of Hemoglobinæmia, Particularly the Paroxysmal Variety.—In most of the cases above enumerated, hemoglobinuria is the result of an obvious or easily demonstrable cause. There is, however, another variety which appears paroxysmally in individuals who are otherwise perfectly well. Its symptoms are extremely characteristic. Although not a very frequent disease, there has been abundant opportunity to study it.

As just intimated, the disease is paroxysmal. Very often an attack is ushered in by frequent and persistent yawning. To this symptom are soon added pain in the limbs, headache, nausea, vomiting, and coolness of the periphery. The temperature speedily rises to 102° F. (39° C.) or more. With this is often associated a decided chill. Sometimes there is a violent pain in the hepatic region. There is a distinct enlargement of the spleen. Then the temperature falls again, perspiration appears, and the patient, although languid and depressed, soon recovers. A slight icteric hue can almost invariably be detected toward the end of the attack, the ordinary duration of which is from two to twelve hours. An eruption of urticaria has been repeatedly seen in connection with the attacks.

The most interesting phenomenon of all remains to be described. We refer to the condition of the urine during and directly after the paroxysm. This secretion presents a dark brownish-red color resembling blood; it may even appear almost black. Its reaction is almost invariably acid; its specific gravity is usually rather low—say 1.008 to 1.012. On boiling the urine, the hemoglobin is decomposed and a brown coagulum of albumen formed. If the fresh urine be examined through a spectroscope, we find the bands *D* and *E* (in the yellow and green) characteristic of hemoglobin, and sometimes also the narrow bands between *C* and *D* indicative of methemoglobin. It is therefore impossible to doubt the existence of hemoglobin in the urine; and yet, upon microscopic examination, we find no red blood corpuscles in the urine, or, in other words, no "hematuria." Frequently there are large numbers of opaque red granules in the urine, the shape of which is extremely irregular. These are, doubtless, granules of hemoglobin. Some of them are free in the urine, some are attached to casts. Of the latter, we find hyaline and a few epithelial casts present. Sometimes masses of hemoglobin assume the appearance of casts. The sediment may also contain a few cells of renal epithelium. The presence of this and of hyaline casts indicates that the kidneys have been slightly irritated by the excretion of hemoglobin. Hence we sometimes observe a persistence of slight albuminuria after the hemoglobinuria has ceased. In mild attacks, also, there may be merely albuminuria, without hemoglobinuria.

If now we examine the blood during a paroxysm, we shall find that hemoglobinæmia is associated with hemoglobinuria of the paroxysmal type, as well as with that occasioned by the action of various poisons. Küssner obtained blood from a patient during a paroxysm, and found that its serum had a ruby-red color, and contained hemoglobin in solution. This proves that the destruction of blood corpuscles takes place within the circulatory system. Indubitable tokens of this destructive process are to be seen upon microscopic examination of the blood during a paroxysm, especially when the paroxysm has been produced artificially in the manner described below. The red blood corpuscles have little tendency to form rouleaux. They are pale, and many of them are irregular in shape (poikilocytes). Irregularly shaped flakes of hemoglobin are also present, and often large numbers of decolorized red blood corpuscles are to be seen. To these latter Ponfick has given the name of "shadows." The exciting cause of the individual attacks of paroxysmal hemoglobinuria is, in most instances, catching cold. A paroxysm is usually due to being outdoors in a cold storm, and perhaps being wet through. In summer the attacks cease completely in patients of this sort; still, as Rosenbach showed by experiment, an attack may even then be artificially excited at any time by exposing the patient's skin on purpose to a decided chill—e. g., by causing the patient to take an ice-cold foot bath, or the like. In order to show that there is merely a local action of cold in these cases, Ehrlich and Boas (each independently) arranged the experiment in the following manner: They tied an elastic ligature firmly around the finger of a subject, and had him immerse it for a quarter of an hour in ice water. In every sample of blood taken from the finger the above-described changes could be most distinctly demonstrated, while blood taken from other parts of the patient's body showed scarcely any abnormality.

But recently doubts have been suggested as to the conclusiveness of this test, as Chvotek found that a simple congestion, such as results from firmly binding a ligature around a finger, is in itself sufficient to cause a decomposition of the red blood corpuscles.

The influence of cold is not the only exciting cause for such attacks, although it is the most frequent one. Paroxysms of hemoglobinuria have been seen after great bodily exertion, particularly after a rather long walk; in women sometimes at the menstrual period, and also after mental excitement, and the like. No explanation has as yet been given why some few human beings have this remarkable sensitiveness of their red blood corpuscles, while most persons undergo the same influences without similar consequences. In this regard should be noted the statement of Murri, that paroxysmal hemoglobinuria is particularly apt to appear in persons who have a syphilitic taint.

Finally, with regard to the origin of the other symptoms of the hemoglobinuric attack, such as fever, at first it was thought that there was a sort of uræmic condition, inasmuch as the urinary tubules are often so choked with granules of hemoglobin that the excretion of urine might well be hindered. Probably, however, another circumstance is still more important—viz., the toxic influence of the fibrin ferment which is formed because of the hemoglobinuria. The hemoglobin, as it is set free, destroys numerous white blood corpuscles, and in this way fibrin ferment is produced, as we have already mentioned.

Another important point is that it is not the kidneys alone which serve for the reception of the constituents of the disintegrated and dissolved blood corpuscles. Ponfick has been led by the result of certain experiments to believe that the spleen and liver are also affected. The spleen appropriates the undissolved remnants of the corpuscles, and, as a consequence, it may undergo considerable enlargement. The liver absorbs a large part of that portion of the hemoglobin which has undergone solution, and converts it into bile. As a result there seems to be an increased secretion of bile. Jaundice is probably due to local biliary stasis and biliary absorption in the liver itself ("hemo-hepatogenous jaundice" of Afanassiew).

Prognosis.—When hemoglobinuria is merely a symptom of other abnormal processes caused by poisoning or by some specific infection, the future of the patient depends entirely upon the severity of the primary disease. An attack of paroxysmal hemoglobinuria would not seem to involve any direct danger to life. Recurrence of the paroxysms is to be expected if the patient be exposed to those influences which produce it. There are no certain means of decreasing the patient's liability to attacks. In a few cases, however, where there had been syphilis, the paroxysms are said to have been permanently banished by mercurial inunctions. Likewise, if we suspect malarial influences, quinin should be tried. But it must be remembered that large doses of quinin may also produce hemoglobinuria in those persons who are susceptible to it (compare the so-called black-water fever of the tropics).

Treatment.—No special treatment is required during the paroxysm itself. The patient must seek refuge as soon as possible from the cold; it is then advisable for him to lie quietly in bed, and to drink a large amount of fluid, so as to wash out the masses of hemoglobin from the kidneys.

CHAPTER VII

SCURVY

(*Scorbutus*)

Prefatory Remarks.—Scurvy is one of a group of diseases which may be termed "hemorrhagic." They all have one predominant symptom—namely, a decided hemorrhagic diathesis, respectively associated in the different diseases with various other more or less pronounced disturbances. This tendency to spontaneous hemorrhage is in many cases, particularly the milder ones, confined more or less exclusively to the skin, but in numerous other instances hemorrhage also takes place into the underlying tissues, such as the muscles or joints, as well as into the mucous and serous membranes.

The distinctions between these various diseases are founded upon the manner in which the hemorrhages occur and the symptoms which attend them. We may mention scorbutus, purpura simplex, purpura hemorrhagica, and peliosis. It should, however, be stated that, although it is possible to distinguish several varieties of disease, each one of which presents a tolerably characteristic picture, there are innumerable transitional forms. It may, indeed, be almost a matter of taste in any particular case what name shall be applied to it. The existence of so many intermediate forms renders it evident that the various members of this group of diseases are at least closely related if not actually identical. We shall even find, upon careful consideration, that certain other diseases not usually regarded as hemorrhagic are nearly akin to the group now under consideration. We refer to certain skin diseases, which are characterized mainly by inflammatory and exudative lesions of the skin. Chief among these should be mentioned erythema exsudativum multiforme, which not very infrequently exhibits some tendency to hemorrhages, and thus presents external appearances closely simulating the forms of purpura. In the last-mentioned forms of disease, however, there appears still another clinical relationship—viz., to the ordinary acute articular rheumatism (*q. v.*). All of the hemorrhagic diseases are distinguished by a frequent involvement of the joints, while, on the other hand, acute articular rheumatism is sometimes associated with hemorrhagic lesions in the skin.

In order to understand the underlying connection between these various disorders, a precise knowledge of their ætiology is requisite. Already considerable evidence has been gathered pointing to the importance of infectious influences in their production (*vide infra*), but no absolute proof has yet been obtained. Still, it must be said that various observations already made indicate that we are likely to find some ætiological relations between the hemorrhagic and the septic diseases. In the meanwhile, we must be guided mainly by the purely clinical phenomena. These, again, indicate that sharp distinctions between the various hemorrhagic diseases would be purely artificial. In this and the following chapters we shall discuss, therefore, merely some of the main types of hemorrhagic disease.

Ætiology.—Scurvy is sometimes sporadic, and sometimes epidemic and endemic in its occurrence. There were formerly very extensive and fatal epidemics of the disease, at a time when the laws of health with regard to large aggregations of human beings were little regarded. The disease was prone to attack armies, or the inhabitants of besieged cities, or, especially, seamen. It was, and to a certain extent is still, one of the diseases most dreaded by the mariner. It has often swept away an entire ship's crew. To-day endemics of scurvy are by no means infrequent, although not so extensive as formerly. They are most apt to occur in prisons and similar institutions and in barracks.

These facts, under the light of our present views in regard to such matters, would almost force us to seek some organic infectious poison as the origin of the disease. Formerly men were inclined to direct their sole attention to such circumstances as the character of the food, the dwelling, the climate, and similar conditions; nor can it indeed be denied that these hygienic factors do exert a decided influence upon the spread of the disease. It is, however, evident that they cannot be its proper cause, for, beyond a doubt, scurvy may occur independently of any of the factors once regarded as essential to its development. These causes must therefore be regarded as simply predisposing influences.

Great ætiological importance has long been ascribed to certain errors in diet. These include the use of bad or insufficient food, the undue predominance of certain kinds of food, and in particular of the salt meats so much employed on shipboard; or, again, the deficiency of certain varieties of food, in particular the lack of vegetable food, and still more of fresh vegetables. Much industry and acuteness have been expended in defending the theory that the lack of vegetable food is injurious because of the deficient supply of potassium salts under such circumstances (Garrod). Nevertheless, this view does not reach the heart of the matter, for in numerous epidemics of scurvy there has been no such lack of vegetable nourishment; and in some instances the diet employed has contained an unusual abundance of potassium compounds.

A like predisposing but not specific influence is exerted by the other factors to which ætiological importance has been assigned. They are indeed often present in epidemic as well as in sporadic cases, but, as previously stated, they may not exist at all. To this class belong damp and unfavorable quarters, cold, moisture, persistent heat, and excessive muscular exertion.

Age and sex exert no great influence upon the disease. Weakly persons seem to be somewhat more liable to be attacked than are the vigorous. The possibility of contagion has been maintained repeatedly, but contagion has not been proved to exist, and unprejudiced observation would incline one strongly to doubt its existence.

[It is certain that scurvy is a disease which we can produce artificially, and that it is preventable in the vast majority of cases. It is now as rare among seamen as it was formerly common—a change which is the result chiefly of care to vary the diet, especially on long voyages. The United States law requires that lime or lemon juice, sugar, and vinegar shall be carried by all sailing vessels bound on ocean voyages or engaged in the fisheries, specifies the circumstances and minimum doses under which the antiscorbutics are to be given, and provides penalties for violation or neglect.]

Clinical History.—The disease does not usually begin suddenly. There is a gradual onset, marked by certain constitutional symptoms. The chief of these are languor and debility, a sense of thoracic oppression, palpitation, and usually a "rheumatic, dragging pain" in the loins and extremities, especially in the lower extremities. The patient is obliged to take to his bed if the case be at all severe, he is very sensitive to cold, and often is drowsy and apathetic. These somewhat indefinite premonitory symptoms last for a few days or a week, when other and more characteristic phenomena appear.

Among these new appearances are spontaneous hemorrhages, occurring chiefly in the lower extremities. There are cutaneous hemorrhages, producing dark red macules of varying size, most of them with a hair follicle in their center, and there are almost invariably hemorrhages into the deeper tissues also. The subcutaneous connective tissue and muscles, and sometimes the periosteum, are affected. These deeper extravasations are a peculiarity of scurvy. They can sometimes be felt as hard, painful swellings in the parts affected, and are sometimes discernible from the discoloration of the skin, which soon results from the solution and diffusion of the blood pigment. The patches present a diffuse bluish color, merging into greenish or yellowish at the periphery, and they are often quite large. They have a precisely similar appearance to "black-and-blue" spots resulting from injury. Of course, the more abundant and the more superficial the extravasation, the more extensive and darker is the macule. Similar appearances may sometimes be observed in the upper extremities and trunk, mainly in severe cases. The face and scalp rarely present ecchymoses.

Sometimes a hemorrhage results in the necrosis and sloughing away of a portion of the skin. The necrosis is succeeded by ulceration ("scorbutic ulcers"). Under unfavorable hygienic influences this process may assume a grave significance. It should also be stated that we may observe other cutaneous disturbances, such as erythema, wheals, vesicles (the contents of which may be tinged with blood—"scorbutic pemphigus"), papules, and pustules. These eruptions are more frequent in some epidemics than in others; they may either be associated with or replace the cutaneous ecchymoses.

In the ordinary sporadic cases of scurvy which occur among us, hemorrhages into the mucous membrane, except of the gums, are very rarely seen. The same is true of hemorrhages from the stomach and other internal organs. In severe cases, during epidemics and under bad hygienic surroundings, it is otherwise; hemorrhages may take place from the nose, stomach, intestines, bronchi, and kidneys, and blood may be effused into the serous membranes.

Next in importance to hemorrhage is another peculiar symptom, presented by the mucous membrane of the mouth, and particularly that of the gums. In order to establish a diagnosis of scurvy in sporadic cases, we must demonstrate the existence of these two main symptoms—namely, the hemorrhage into the skin or muscles, and the changes in the gums now to be described.

The scorbutic changes in the gums usually appear quite early in the course of the disease, being in many cases simultaneous with the hemorrhages, although they may either precede or follow the latter. The gums assume a bluish hue, become swollen and spongy, are painful, and have a tendency to bleed. The changes are usually most pronounced in the salient parts of the gums between the teeth. It is a remarkable fact that they are hardly visible at all at places where there are no teeth; and the gums of very young children and of aged patients remain almost intact. In severe cases the gums are not only swollen but necrosed; the change is at first a superficial one, but it may extend inward and produce dirty-looking ulcers. Other parts of the mouth are liable to become involved in the ulceration, producing a diffuse ulcerative stomatitis, and giving the breath a most offensive odor.

Certain other local and constitutional phenomena are not infrequent, though less characteristic than the hemorrhages and the alterations in the gums. Chief among the general disturbances is scorbutic anæmia. This is often referable in part to the unfavorable hygienic influences surrounding the patient, but the disease itself seems to impair the general nutrition. The patient looks pale, has a dry skin, and loses flesh rapidly. The temperature is often normal. Sometimes there may be an occasional rise of temperature, either in the beginning or in the further course of the disease. If complications occur, they are not infrequently accompanied by considerable fever.

Among more localized symptoms should be mentioned the premonitory sore throat which sometimes occurs. It is usually of the ordinary catarrhal variety, but it may assume a hemorrhagic character. Bronchitis also may occur. Lobular pneumonia and genuine lobar pneumonia have been repeatedly seen in severe cases. Pleurisy, pericarditis, and inflammations of other serous membranes occasionally complicate the disease. They may likewise display a hemorrhagic tendency in the exudations to which they give rise. Disturbances in the joints are sometimes seen, and are characterized by an effusion of liquid into the articular cavities, which effusion may be either serous or hemorrhagic. This is a favorable opportunity to call attention to a peculiarity common to all the hemorrhagic diseases and allied affections (*vide supra*)—they are apt to be associated with articular swelling.

The pulse may be somewhat accelerated, or may be slower than normal. It is usually small and compressible. Endocarditis may occur, but it is very exceptional. The blood does not present any constant and characteristic alterations in scurvy. The spleen may be decidedly enlarged, particularly in severe cases. Albuminuria has been repeatedly observed, but it is almost wholly confined to severe cases, in which, indeed, a typical acute nephritis may be developed.

Course of the Disease. Varieties of Scurvy. Prognosis.—The sporadic cases usually met with in this region almost invariably pursue a favorable course. The symptoms are confined mainly to the constitutional disturbance, ecchymoses in the lower extremities, and the affection of the gums, the grave complications above mentioned being rarely met with. The average duration of the disease is, nevertheless, some weeks. Recovery is deferred in proportion as the hygienic surroundings are bad, but even then the termination is almost sure to be favorable. Scurvy has, however, a noteworthy tendency to relapse, so that the course of the disease may be very much prolonged. Even when the illness has terminated in recovery, there remains behind a certain liability to fresh attacks of scurvy, just as acute articular rheumatism is apt to recur. We were acquainted with a patient in Erlangen who was severely ill with scurvy some five or six times within a period of a few years, and this without any demonstrable cause.

The prognosis in grave cases, occurring under favorable hygienic influences, and aggravated by the lack of proper food and attention, is far otherwise. Here death is not infrequent, sometimes as a result of progressive cachexia, sometimes because of pneumonia, pericarditis, cerebral hemorrhage, or a similar intercurrent disease.

Anomalous or rudimentary cases of scorbutus may occur. They are most apt to be seen when the disease is epidemic or endemic. As a rule, the symptoms are mild. We find, for example, a scorbutic gingivitis and stomatitis without hemorrhage, or, on the other hand, hemorrhages into the skin and mucous membranes unattended by alteration in the gums. There have even been cases reported of simple scorbutic anæmia without any local symptoms.

[The experience of army surgeons during our Civil War deserves mention in this connection. Hammond, Woodward, and others state that many cases classed in the sick reports as "general debility" were cases of incipient or imperfectly developed scurvy, hemorrhage from mucous membranes or into the skin being absent.]

Diagnosis.—The diagnosis of scorbutus is almost self-evident when the two chief symptoms of hemorrhage and alteration in the gums are both present. If, however, one or the other of these symptoms is suppressed or imperfectly developed, it may be difficult to determine what disease we have before us, or to exclude ordinary stomatitis, rheumatic peliosis, and similar diseases. One characteristic sign should be emphasized again in this connection. The hemorrhage of scurvy is scarcely ever confined to the skin, but almost invariably there are large extravasations deeper in, in the muscles and fasciæ. These occasion, secondarily, the characteristic bruise-like bluish-yellow spots seen through the skin.

Treatment.—The essential requisites in the treatment of scurvy are proper hygiene and diet. Fresh air, suitable nourishment, and good nursing, if promptly supplied, are usually of themselves sufficient to induce recovery, while the physician possesses no remedies which compensate for their absence.

The belief that a main cause of scurvy lies in a deficiency of fresh vegetables has given rise to a practice, still in vogue, of prescribing a great abundance of fresh vegetables, such as lettuce, spinach, and sorrel, fruit, lemonade, and other drinks prepared from fruit sirups. There is no reason to deviate from a course to which experience has given its sanction, although we have repeatedly had opportunity to see that the administration of fresh vegetables is by no means essential to rapid recovery. Patients supplied with any other proper nourishment thrive equally well. There is still scientific basis for the reputation certain varieties of plants have attained as "anti-scorbutics," such as the spoonwort (*Cochlearia officinalis*), so frequently mentioned in accounts of early polar expeditions. The administration of vegetable acids and the salts of potassium (bitartrate and nitrate of potassium), in a chemically pure form, has also been repeatedly tried, but it has not gained popularity.

The drugs most employed are the bitters and tonics. They have no specific value, but are perhaps as good remedies to prescribe as any. We may give a decoction of cinchona, ʒijss. in ℥v (gm. 10 to 150) with ʒss. (2 c.c.) of dilute sulphuric acid and ʒv (20 c.c.) of raspberry sirup, or some preparation of gentian or a similar bitter. It was once believed that the internal administration of the mineral acids exerted a specially favorable influence upon the hemorrhagic diathesis; but this is very doubtful.

Certain symptoms may demand attention; in particular, the affection of the mouth and gums. It is of great importance to cleanse the mouth frequently with disinfectants and mild astringents, such as chlorate of potassium or sage tea. It is also advisable to paint the inflamed and spongy gums at short intervals with tincture of myrrh or tincture of rhatany. The absorption of the ecchymoses in the lower extremities will be promoted by cautious massage. Rubbing with linimentum chloroformi and the like gives relief from the pain caused by the extravasations into the deeper tissues. In severe cases stimulants are often demanded, such as camphor, ether, and alcohol. Such complications as appear may also demand special treatment.

Convalescence is promoted by continued attention to diet, bathing, and the administration of iron and quinin.

APPENDIX

BARLOW'S DISEASE

In this connection we desire to mention briefly a peculiar symptom-complex which has been classed with scurvy, in regard to the real nature of which, however, we do not yet possess any definite knowledge. The disease is, therefore, usually named after the person who discovered it (1883), as Barlow's disease.

The disease has been seen thus far in children alone, and most often between the ages of six and eighteen months; almost all the children had previously shown definite signs of rickets. There is little, if any, fever, but an extremely painful swelling of the bones, particularly of the epiphyses of the long bones, though the lower jaw, cranium, and other parts may be involved. The epiphyses may become loosened, but the joints themselves are not affected. Upon careful examination it is found that the swelling is due to subperiosteal hemorrhages.

In consequence of these changes in the bones, the movements of the child are naturally seriously impeded. The child screams on being touched and when it tries to move. Finally, the legs come to lie almost immobile in bed. In association with these there may be other indications of a hemorrhagic diathesis, such as hemorrhage into the skin and into the serous membranes, and hematuria. Distinct scorbutic lesions show themselves in those portions of the gums where the child has already cut his teeth. Marked anæmia generally sets in. In severe cases the child acquires a waxy pallor. Such complications as bronchitis, broncho-pneumonia, gastric catarrh, etc., may occur. The disease usually takes a chronic course, unless the only proper dietetic treatment (*vide infra*) is promptly applied. In milder cases, which are the rule, the extravasations of blood into the bones are absorbed and do not recur, and after some weeks, or perhaps months, complete recovery ensues. In more severe cases, however, the hemorrhages constantly recur, and the child sometimes dies finally of exhaustion.

The prognosis of the disease is favorable, since we know that its cause is to be sought in certain not yet understood anomalies of nourishment. The disease appears only in children who are artificially nourished, and particularly those who are fed on boiled milk. Consequently, the treatment must consist, above all, in special dietetic measures. Of course, human milk would be best, but if this cannot be procured, the child should be given raw cow's milk instead of sterilized boiled milk. Older children should receive in addition weak beef broth or two or three teaspoonfuls of raw meat juice (*succus carnis recente expressus*), also vegetables (spinach, carrots) and fruit juices (raspberry, lemon [orange] currant). Large-sized hemorrhages may require incision and the emptying out of the blood clot. In most cases, however, lead-water compresses and light massage will suffice. If loosening of the epiphyses sets in, the application of a firm bandage becomes necessary.

CHAPTER VIII

PURPURA. MORBUS MACULOSUS WERLHOFII. PELIOSIS

(*Purpura hemorrhagica*)

As already stated in the preceding chapter, the various "hemorrhagic diseases" are so intimately related to one another that it is quite impossible to make a rigid categorical division of them.

From a clinical standpoint this fact is the all-important one—namely, that there are cases in which the foremost symptom is the spontaneous occurrence of hemorrhage. There are cutaneous ecchymoses, and there may be at the same time hemorrhages in the internal organs and into the mucous membranes. In the milder cases of this sort these hemorrhages constitute almost the only symptom of disease; but they may be associated with considerable general disturbance, indicated by fever and weakness, or with certain local complications. The true cause of these diseases has not yet been discovered. There is seldom any evidence of an exciting cause, and the disorder may attack either the well nourished or the poorly nourished, the old or the young, men or women. There is, however, an indisputable relationship between these diseases and certain others—namely, scurvy, erythema exudativum, and perhaps acute rheumatism and endocarditis. This similarity indicates that the process is of an infectious, or toxic, character. Such an assumption promotes greatly a proper understanding of the phenomena under consideration. It is also said that subcutaneous injections of blood from patients with morbus maculosus may cause a like affection in rabbits (Petrone and others). In some few cases the weight of evidence would seem, however, to point to an antecedent impairment of nutrition in the walls of the blood vessels. A good example of this is seen where cutaneous ecchymoses occur in old and marantic individuals (*peliosis senilis*). There is some doubt whether these exceptional cases belong with the others.

The mildest forms of the diseases under discussion are termed *purpura*. The hemorrhages are seen mainly in the skin of the lower extremities, and are apt to take place in the follicles. There may also be ecchymoses upon the trunk and upper extremities, but the mucous membranes and the deeper tissues remain intact. A means of distinguishing purpura from scorbutus lies in the fact that in purpura there are no hemorrhages into the muscles and no lesions of the gums, although it should be confessed that transitional forms between the two occur. The disorder is called *purpura simplex* if the cutaneous ecchymoses constitute the only symptom, or, at any rate, the only important one. These cases almost invariably terminate in recovery, and are over at the end of ten days or three weeks. Sometimes elevations of the skin are formed resembling wheals, and hemorrhages take place here and there into them. This subvariety has been called by some *purpura urticans*. It forms a connecting link between purpura simplex and those cases of erythema exudativum which are associated with hemorrhage. Further particulars may be found in special works upon dermatology.

Quite often the hemorrhages are attended by "dragging rheumatic pains"; such cases are termed *purpura rheumatica* or *rheumatic peliosis* (Schönlein). There may also be constitutional disturbance, slight fever, anorexia, and indisposition to either bodily or mental exertion. There may sometimes be actual arthritis, with an inflammatory effusion into the joints. The knee and other joints of the lower extremities are most apt to suffer in this way. The gums are usually normal; nor is there, as a rule, hemorrhage into the mucous membranes or the viscera. These cases may last but two or three weeks. Often, however, they are more tedious, being marked by the recurrence of the ecchymoses and articular pain. Most of them get well at last.

No sharp dividing line can be drawn between the forms of purpura thus far described and certain graver cases. These latter are most of them grouped under the name of *purpura hemorrhagica,* or its preferable, because more distinctive, synonymn—every purpura being hemorrhagic—*morbus maculosus Werlhofii.* The cutaneous ecchymoses in this class of cases are usually extensive; and, furthermore, we have hemorrhages into the mucous membranes of the nose, mouth, soft palate, stomach, and intestinal canal, as well as into internal organs (the brain and kidneys), and also into the serous membranes. The constitutional disturbance is apt to be severe. The condition may be distinctly "typhoidal." Fever may be entirely absent, even in grave cases, although sometimes there is a considerable rise in temperature.

There are usually no local symptoms beyond those already mentioned. In typical cases the gums remain intact. Swelling of one or more joints has been repeatedly observed, as have also endocarditis and acute hemorrhagic nephritis. If marked cerebral symptoms are developed, suggesting an apoplectic shock, we may surmise that a cerebral hemorrhage has taken place. It should also be stated that marked gastrointestinal disturbance may occur. Cases of this sort have been observed by Henoch in children. They may also occur in adults. In rare instances there may be intestinal ulceration, with perforation and consequent peritonitis. The spleen may undergo acute enlargement.

All the symptoms which have been enumerated render distinct the similarity of the clinical picture, in severe cases of morbus maculosus Werlhofii, to that of severe septic infection. In fact, it is not impossible that continued investigation will deprive morbus maculosus of its individuality as a specific form of disease, and show that it is merely a special form of profound sepsis.

The prognosis in purpura hemorrhagica should always be a guarded one; the patient is in danger both from the general depression and anæmia and from certain special lesions. Even a severe case may, however, recover. The disease sometimes proves very tedious; it may occupy several months.

Treatment.—The general regimen to be prescribed is similar to that directed in scurvy. The physician must strive to support his patient's strength by proper nourishment. A great many internal remedies have been recommended, most of them on purely theoretic grounds. It is difficult to say whether they actually exert a favorable influence upon the course of the disease. The following drugs are chiefly employed: Ergotin, perchlorid of iron, dilute sulphuric acid, and cinchona. If there be swelling of the joints or endocarditis, we should advise a trial of salicylic acid or antipyrin. Such special symptoms as demand attention, particularly heart failure, should be treated according to general principles.

CHAPTER IX

HEMOPHILIA

Definition and Ætiology.—Hemophilia is the term used to denote a peculiar constitutional anomaly, exhibited in a remarkable tendency to spontaneous and traumatic hemorrhage. The condition is probably in every instance congenital, and is usually hereditary; the existence of families of "bleeders" has long been known. Generation after generation displays frequent cases of hemophilia, both among the direct descendants and the lateral branches. Bleeders are very apt to have a numerous progeny. Not all of the children, however, fall victims to the disease. Grandidier has pointed out two facts which are of interest in this connection, as they might aid in deciding as to the marriageability of certain persons.

If a man belonging to a family of bleeders marries a healthy woman, neither a bleeder herself nor inheriting a predisposition to hemophilia, his children are almost certain to be healthy, even though the father himself be a bleeder. On the other hand, a woman belonging to a family of bleeders, even though she herself be healthy, will almost always have some children who are subject to hemophilia. In other words, hereditary predisposition is transmitted much oftener through the female than through the male members of the family. Hemophilia itself is, on the contrary, much more frequent in the male sex than in the female; at least this is true of the pronounced cases. Hössli, who has lately published a very careful family tree of bleeders of Tenna (Canton Graubünden), comes to the following conclusions: "The inheritance of hemophilia is often from the father, through the daughter, to the grandson; also from the mother, through the daughter, to the grandson, and most rarely directly from the father to the son." It is doubtful whether race and place of residence are of ætiological importance. So far as is known, hemophilia appears to occur in all countries, although it is fortunately rare everywhere.

[A similar transmission through the females, who themselves usually escape, is seen in color blindness and in pseudo-hypertrophic paralysis.]

The real causes of hemophilia are entirely unknown to us. We can make one or two steps toward finding the source of the hemorrhage, but we are unable to proceed further. It would seem that the bleeding must depend, in the first place, upon an abnormal delicacy of the walls of the vessels predisposing them to rupture, and, secondly, upon deficient coagulability of the blood. This latter abnormality is evident from the fact that in hemophilia it is difficult to check even the most insignificant hemorrhage. Thus far all attempts to discover any anatomical or chemical explanation of this imperfect coagulability have been vain. It has not been possible to detect any variation in the saline constituents of the blood, or in the amount of albuminoids, such as fibrinogen, that it contains, or in its morphological elements. And likewise no anatomical change in the vascular walls or the heart has yet been reported which throws light upon the character of the disease. Various authorities have laid stress upon the small diameter of the arteries and the thinness of the intima, but these conditions may occur independently of hemophilia. Fatty degeneration of the intima is, to be sure, often found in connection with this disease, but it is doubtless rather a result of the coincident anæmia than the cause of the hemophilia. The observations with regard to the heart are very contradictory; sometimes it is found to be very small, sometimes of normal size, and again actually hypertrophied.

The subjects of hemophilia do not present any distinctive constitutional peculiarities. It has been stated that they are very apt to be blonds with a delicate white skin, and superficial and abnormally distended cutaneous veins, but the exceptions to this rule are not a few.

Clinical History.—Hemophilia does not display equal malignity in all cases. If we have opportunity to obtain thorough information with regard to families of bleeders, we shall find that quite often rudimentary varieties occur side by side with typical and severe cases. There is, to be sure, a striking tendency to hemorrhage even in them, but the hemorrhage never assumes threatening proportions. By perseverance and industry it is possible to collect an almost unbroken series of cases, varying in degree from extreme mildness to extreme severity. The following sketch applies mainly to typical and severe cases.

That hemophilia is an hereditary constitutional disease is shown by the fact that it sometimes appears in earliest infancy. Many cases of umbilical hemorrhage in the newborn are referable to hemophilia. Of course this does not apply to all cases. In Jewish children the disease may betray itself for the first time when the rite of circumcision is performed. In many cases the disease is not recognized at so early a period; but this is no proof that the disease is not already developed, inasmuch as the young child is not much exposed to traumatism and other causes which naturally occasion hemorrhage.

The most striking symptom in a fully developed case of hemophilia is the occurrence of severe hemorrhage as a result of the most insignificant causes. A slight blow produces a " black-and-blue spot " such as is ordinarily seen only after a very violent injury. The prick of a pin, a slight cut on the finger, or the extraction of a tooth, may give rise in hemophilia to an obstinate and alarming hemorrhage. Epistaxis may be caused by blowing the nose, hemorrhage from the gums by brushing the teeth, and so on. Whether there is ever a perfectly spontaneous hemorrhage is uncertain. It is true that in severe cases hemorrhages take place independently of any visible cause. This may be seen in the skin and mucous membranes (nose and gums), and in rare instances we may even have free hemorrhage from the stomach, intestines, or urinary passages. Yet it may be doubted whether these occurrences are not the result of comparatively insignificant mechanical injuries which escape our notice. At any rate we scarcely ever find hemorrhage taking place into the parenchyma of the viscera in places where injury from external sources is entirely out of the question. This fact constitutes an important point of distinction between hemophilia and the acquired hemorrhagic diathesis.

The second important symptom of hemophilia has been already referred to; it is extremely difficult, and may even be impossible, to check by artificial means any free hemorrhage which may occur. It is this which makes the disease so dangerous, and prevents most patients from reaching old age. It has frequently happened that an apparently trifling wound of the skin, some insignificant operation, or a leech bite, or in women childbirth, has started up a hemorrhage which eventually became fatal. In other cases the hemorrhage is finally checked, but not until it has caused profound anæmia. Bleeders are apt to recover with remarkable rapidity from the effects of excessive hemorrhage, yet continually repeated hemorrhages may lead to a persistent and profound anæmia, attended by all the symptoms described in the preceding chapters.

We see, therefore, that the general condition in hemophilia varies with the severity of the individual case, and with the more or less fortuitous circumstances which develop its dormant characteristics. If no special accident occurs, the patient may maintain the appearance of perfect health for years. In the worst cases, however, such a state is very temporary, if it exists at all, because the hemorrhages can be so easily excited. As a consequence the skin almost always presents a greater or less number of ecchymoses, while hemorrhages from the internal organs contribute from time to time to the general debility and anæmia. The manner in which the whole picture of the disease may be dominated by the two last-mentioned symptoms need not be further specified. Certain complications may occur in hemophilia, but they are little characteristic. There is a noticeable tendency to "rheumatic" inflammation of the muscles and swelling of the joints, wherein is seen a striking analogy to the "hemorrhagic diseases." Often there is an actual effusion of blood into the joints. This may cause considerable functional disturbance of the joints, and eventuate in anchylosis. Various writers have also called attention to the comparative frequency of neuralgia, especially in the trigeminus.

Prognosis.—In only too many instances the victims of hemophilia die in childhood; in other cases the patient attains an advanced age. A fact of great practical importance is that often, although not invariably, hemophilia grows gradually milder with advancing years. If, therefore, the patient have survived the period of adolescence, we may believe that his prospects are gradually improving. The prognosis of hemophilia is obvious. The amount of danger at any given time depends upon the severity of the hemorrhage and the consequent anæmia. The comparative severity of the case must be judged from its previous history; as has just been said, the prognosis grows more favorable as the patient grows older.

Treatment.—Prophylaxis assumes a very important place in the treatment of hemophilia. First, children who inherit a tendency to the disease, or who have given evidence of its existence, should be treated with a view to improve their general constitutional condition, so as to check the development of the disease as far as possible. The means to this end need not be described at length. They comprise good nourishment, fresh air, cautious endeavors to harden the system, baths, and tonics. Secondly, when hemophilia already exists, the patient should be guarded as much as possible from any mechanical injury, such as might excite hemorrhage. Thus, caution is demanded in performing vaccination and operations.

As regards direct treatment of the disease, no effectual remedy is known. The administration of ergotin, acetate of lead, and similar drugs is indicated, if at all, only when hemorrhage is actually taking place, and even then it is very apt to fail. According to certain interesting, more recent observations, the injection of normal blood serum [or of diphtheritic serum] seems to offer more hope of success. The local application of blood serum to the bleeding surface has also been tried repeatedly with success. The description of the surgical measures for the control of hemorrhage need not be described here. They do not differ essentially from those employed when hemorrhage occurs independently of hemophilia.

CHAPTER X

DIABETES MELLITUS

Definition and Ætiology.—Under normal circumstances the blood always contains a slight amount of grape sugar (glucose, dextrose), which, with the greatest variations of diet, remains about the same (0.7 to 1 per cent); but this ingredient does not usually pass over in appreciable quantities into the urinary excretion. But the sugar formed from the carbohydrates and that ingested as such and absorbed by the portal vessels, as also the glycogen formed from the proteids and, under certain circumstances, even from the fat, are stored up in the glycogen depots in the liver and muscles. From this glycogen reservoir only so much enters the blood as the body requires for muscular strength and heat. Only when, for any reason, this glycogen depot in the liver is defective, either because it is overfilled or is unable to retain its glycogen reserve, does grape sugar enter the blood in increased amounts. To get rid of the harmful excess, the body excretes a portion of the grape sugar in the urine. As a result of the abnormally great glycæmia, glycosuria develops. This is seen as a more or less temporary phenomenon under the most varied conditions. The amount of sugar in the urine is usually comparatively slight, and it soon disappears again. Its presence does not imply any persistent abnormal condition. This phenomenon has been termed glycosuria or melituria, in contrast with the peculiar disease which has for its chief symptom a persistence of sugar in the urine, and has therefore received the name of diabetes mellitus.

It is not necessary in this connection to enumerate all the causes of temporary glycosuria. It may be briefly mentioned, however, that glycosuria is observed even in healthy persons after excessive indulgence in sugar. If a healthy person takes at one time about 7 ounces (gm. 200) or more of grape sugar dissolved in water, there will probably be after an hour or two a demonstrable, although not a very great, amount of sugar in the urine. This is the so-called normal alimentary glycosuria. In this case the liver and the blood are so overburdened with sugar that the latter can neither be stored nor oxygenated fast enough. A portion of the sugar, therefore, escapes change, and is excreted with the urine. If the same amount of sugar is given in divided doses at intervals, there will be no glycosuria.

Therefore, in healthy individuals, even after a most liberal amylaceous diet, glycosuria never develops. The conversion of starch into sugar does not take place so rapidly that an overflooding of the blood with sugar is possible. If glycosuria appears upon the administration of small amounts of sugar (2 to 3 ounces [gm. 50 to 100]), we are justified in speaking of a morbid alimentary glycosuria, the power of the body to store and dispose of sugar being subnormal. An abnormal glycosuria of this sort is observed, for example, not infrequently in obese beer drinkers and in patients with severe traumatic neuroses, or with exophthalmic goiter. Temporary glycosuria is observed not infrequently also without an especially liberal sugar ingestion in certain kinds of poisoning, particularly after severe poisoning with carbonic oxid, morphin, hydrocyanic acid, mercury, nitrite of amyl, and curare. Temporary glycosuria has also been seen occasionally in connection with the acute infectious diseases —for example, malignant pustule, cholera, typhus or typhoid fever, scarlet fever, diphtheria, and malarial poisoning. A far more frequent cause is disturbance of the nervous system. Thus, glycosuria may result from severe concussion of the brain, fracture of the skull, cerebral hemorrhage, cerebrospinal meningitis, and after epileptic fits. Glycosuria sometimes appears also in association with exophthalmic goiter, and acromegaly (*q. v.*) is strikingly often complicated by a permanent profuse glycosuria. It is especially apt to occur when there is disease of the medulla; and we need hardly point out how close is the connection between this clinical fact and the famous discovery of Claude Bernard, who found that, when a definite experimental injury is inflicted upon the floor of the fourth ventricle, glycosuria inevitably follows. This glycosuria is explained by assuming that, as a result of nervous influences upon the circulatory conditions of the liver, or perhaps also upon the liver cells themselves, the liver discharges its supply of glycogen into the blood in the form of grape sugar. The blood is overwhelmed with the sugar, and a portion escapes into the urine.

In some other organic diseases, also, there may be a temporary and symptomatic glycosuria. Formerly, special attention in this regard was directed to hepatic diseases, but it is to be noted that, as a rule, even when there is extensive disease of the liver—for example, in hepatic cirrhosis—no glycosuria is observed, even if the patient is given large amounts of sugar in his dietary. On the other hand, the combination of persistent diabetes mellitus and hepatic cirrhosis is not very exceptional. As we shall point out again later on, we regard both diseases in these cases as coördinate effects of the same cause, chronic alcoholism. We shall likewise refer again to the relation between diabetes and renal disease.

This question has assumed particular interest since von Mehring has discovered in phloridzin, a glucosid in the bark of the roots of apple and cherry trees, a substance which causes a very large amount of sugar in the urine if given to dogs, rabbits, etc. A marked glycosuria may be produced in man also by giving phloridzin, without other disturbances of the general condition. In phloridzin diabetes the sugar content of the blood is not increased, but diminished, and it is therefore not improbable that phloridzin glycosuria is different from the other toxic glycosurias, in that it depends upon a change in the renal epithelium, as a consequence of which the cells become permeable to the sugar which is circulating in the blood.

Especial interest attaches to the question of the relation between glycosuria and diseases of the pancreas. Minkowski and von Mehring have made the important discovery that we can excite severe diabetes in dogs by extirpation of the pancreas. The ligation of the excretory duct of the gland, or the diversion of the pancreatic juice into a fistula leading to the surface of the body, does not excite diabetes, nor does the diabetes occur if the pancreas is partially and not wholly extirpated. Pancreatic diabetes, therefore, implies a disturbance of a special function of the pancreas, of which we have as yet no intimate knowledge, but the abolition of which prevents the complete splitting up or oxidation of sugar in the normal organism. The deposit of glycogen in the liver and muscles is also inhibited. In animals without a pancreas, even after a liberal amylaceous diet, only the slightest traces of glycogen can be found in these organs.

Diseases of the pancreas in man (carcinoma; pancreatic calculi, with secondary atrophy of the gland) occasion glycosuria sometimes, but by no means invariably. The atrophy of the pancreas observed in many cases of genuine diabetes will be discussed later on.

Finally, we would briefly refer to the fact that under certain conditions other forms of sugar besides dextrose may be found in the urine. In pregnant and puerperal women, with a profuse secretion and an insufficient discharge of milk, milk-sugar not infrequently enters the blood, and then appears in the urine (so-called lactosuria). Milk-sugar gives the reduction reactions, and is dextrorotatory, but is not fermented by yeast. In isolated cases, in otherwise healthy individuals, pentose ($C_5H_{10}O_5$) has also been found in the urine without simultaneous glycosuria. Pentose likewise gives the reduction reaction, but does not ferment and is nonrotatory. Pentosuria is of a certain practical importance, as it may lead to confusion with real diabetes. It sometimes occurs, strange to say, as a family affection.

In contrast with the temporary phenomenon of glycosuria, we apply the name diabetes mellitus to a chronic morbid state, in which a permanent disturbance in the metabolism of the carbohydrates is indicated by a persistent excretion of sugar in the urine, and in which this anomaly of metabolism is the main symptom of the disease. If the excretion of sugar in this disease is not absolutely persistent, at least it is sure to occur under certain conditions of nutrition. It is doubtful whether diabetes mellitus is to be regarded from an ætiological standpoint as a single, distinct disease. With regard to most of the "pure" cases, particularly the severe form of diabetes which usually occurs in youthful individuals without any demonstrable cause and also without any primary organic lesion, there is great probability in the assumption that the morbid process is a single and definite one, although we are as yet unacquainted with its real nature. On the other hand, with regard to the so-called milder forms of diabetes mellitus, particularly those which occur in middle and advanced life, there are probably various causes which may excite the disease, and which, evidently, in the end all have an effect upon the carbohydrate metabolism. Still, it must be emphatically stated that no sharp dividing line can be drawn between the "severe" and the "milder" forms of diabetes.

Even if the actual cause of diabetes is entirely unknown to us, we are nevertheless acquainted with certain circumstances which favor the development of the disease. These causative factors are the following: first, heredity; diabetes has not infrequently been observed to occur in several generations of the same family, or in several brothers and sisters. Other anomalies of metabolism, such as gout, the uric-acid diathesis, obesity, and arteriosclerosis, are met with strikingly often in families of diabetics. The disease may also occur in families where there is a hereditary predisposition to nervous diseases. For example, we observed once in the same patient the remarkable combination of diabetes and progressive muscular atrophy of spinal origin. Second, improper mode of life; by this is meant chiefly unsuitable diet, especially the persistent overindulgence in starchy foods and sugar; sedentary habits are also considered harmful, especially if associated with overeating. This is said to be the reason why diabetes is more frequent among the wealthy classes, and why it is comparatively common in corpulent persons (*vide infra*). Our personal observations lead us to ascribe to long-continued and excessive beer drinking (four or five liters and more per day) an influence upon the development of diabetes. In such cases it may be that we have combined action of the excessive ingestion of carbohydrates in a state of solution, and of chronic alcoholism. It can, in our opinion, be no mere coincidence that diabetes so often attacks brewers, innkeepers, and the like, in Bavaria. Furthermore, the complication of diabetes with other results of chronic alcoholism, such as hepatic cirrhosis, chronic nephritis, and alcoholic neuritis, is an argument for the correctness of our supposition. The "diabetes of obesity" has been long recognized, but in many cases it is really a "beer diabetes." Third, taking cold and getting wet are said, in occasional rare instances, to determine the appearance of diabetes. The author has never seen a clear case of this sort. Fourth, emotional disturbances, excessive mental exertion, anxiety, and passion, are sometimes thought to occasion the disease, and apparently with some reason. We know of several cases of diabetes where the first symptoms followed immediately after extreme mental excitement. Fifth, it is very remarkable that sometimes the same factors which we have already seen to be possible causes of temporary glycosuria may also occasion a chronic diabetes mellitus; thus, cases of diabetes have been known to follow injuries to the head, and such acute infectious diseases as typhus, typhoid, and scarlet fevers, cholera, and malarial poisoning. Sixth, a few authors (Schmitz and others) have hinted at the possibility of the disease being transferred by contagion—for example, in the case of married people. This interpretation of the scattered instances which have been reported is, however, at least open to doubt. Seventh, a very important matter is the occurrence of diabetes in association with, or as a sequel to, certain other constitutional diseases. In this category belong the above-mentioned diabetes of the obese; also the simultaneous appearance of diabetes and genuine gout (*q. v.*); and finally, the extremely important combination of diabetes with general arteriosclerosis. That all these combinations do frequently occur cannot be doubted, but it is very difficult to explain the connection between them; often both conditions may be the simultaneous effect of the same cause—for instance, congenital anomalies of constitution, unhygienic modes of life, and alcoholism. In other cases, however, all these anomalies of nutrition point to an abnormal constitutional diathesis as a common ætiological factor. It appears to us particularly worthy of mention that not infrequently, as has already been stated, we observe the occurrence in the same family of such obviously allied constitutional diseases as obesity, gout, diabetes, and arteriosclerosis.

If we survey all the facts thus far known with regard to the ætiology of diabetes, we feel obliged to conclude, just as in almost all other constitutional diseases, that in regard to the development of diabetes we must distinguish endogenous and exogenous factors. The endogenous relate to the constitutional tendencies which the patient possesses, perhaps by inheritance; and the exogenous to harmful influences from without, such as trauma, infection, and intoxication. The severe cases of diabetes which seem to occur absolutely spontaneously in young persons, are mainly of an endogenous character, while in the milder forms of later years the above-mentioned exogenous influences often play an important part, although even in them the endogenous factor cannot be entirely disregarded, for the same noxious influence does not produce, by any means, the same effect upon all persons.

Diabetes occurs everywhere, but certain countries and districts seem to be particularly liable to it—for example, India, Ceylon, and Italy. In Germany, Würtemberg and Thüringen are said to present the largest relative number of cases. We have, however, seen so many cases of diabetes in Bavaria, and later in Silesia, that it appears to us probable that the disease is pretty nearly equally distributed all over Germany. Hebrews are very liable to the disease. Most cases occur in patients between thirty and forty years of age. Next in liability to the disease come younger individuals, under thirty and over twenty years old. After the fiftieth year diabetes is not exceptional, but, in the other direction, children under ten are very seldom attacked by it, although they are not absolutely exempt. Diabetes in youth and childhood is almost always of the severer type. With regard to sex, males are much more often attacked than females.

Clinical History.—With few exceptions, the symptoms of diabetes mellitus come on slowly and gradually. Sometimes the symptoms are merely general and indefinite, such as languor, emaciation, weakness, and deficient endurance. Sometimes we have mild nervous disturbances, including headache, mental depression, wakefulness, and neuralgia, and in still other cases gastro-intestinal symptoms, including nausea, eructations, and irregularity of the bowels. At last the patient's attention is called to the altered character of the urine, and particularly to its increased amount. He also notices that he is very thirsty, and that, in spite of his enormous appetite, he is constantly growing weaker. Sometimes quite different symptoms first arouse suspicion of the existence of diabetes; these will be mentioned later. In order to make a diagnosis of diabetes mellitus, a knowledge of the abnormal character of the urine is indispensable, so that it is proper to begin our consideration of the symptoms of diabetes with a description of diabetic urine.

1. CHARACTER OF THE URINE. DEMONSTRATION OF SUGAR.—Usually the first point that attracts attention is the increased amount of urine. There are often 100 to 150 ounces (3 to 5 quarts, 3,000 to 5,000 c.c.) excreted in twenty-four hours, and sometimes the amount equals or exceeds 10 or 12 quarts (8,000 to 12,000 c.c.), Under suitable treatment and with proper diet the amount may, of course, be much smaller. In not very exceptional cases, mostly of the milder varieties (*vide infra*), there is slight, if any, increase in the amount of urine (*diabetes decipiens*); and in severe cases with initial polyuria we may often observe that during intercurrent diseases, and likewise in the last days before the fatal termination of the disease, the daily amount of urine diminishes.

In color the urine is light yellow, corresponding to its amount. It often has something of a greenish hue, but a small quantity of it may seem almost as colorless as water. Ordinarily the urine is clear and without sediment; but after it has stood for some time it may become cloudy, usually as a result of the abundant development of fermentation spores.

The odor may be somewhat aromatic, suggesting acetone (*vide infra*). Its taste is said sometimes to be distinctly sweetish. The reaction is acid, and the acidity of the urine may increase on standing, because of the alcoholic and lactic-acid fermentation processes which the sugar undergoes.

The specific gravity is almost invariably greatly increased, as a result of the large amount of sugar. If a pale urine is found to have a specific gravity of more than 1.025, we may feel almost certain that it contains sugar. Specimens often have a specific gravity of 1.030 to 1.045, and even higher. In exceptional instances the specific gravity may fall below 1.020. A specific gravity below 1.020 is exceptional, but one should never be kept from making an examination for sugar because of the low specific gravity of the specimen. We have ourselves demonstrated sugar by means of the fermentation test in urine which had a specific gravity of 1.007.

The diagnosis requires that sugar be detected in the urine. The sugar found in both the blood and the urine of diabetic patients is, as already mentioned, grape sugar (glucose, dextrose, $C_6H_{12}O_6$). The amount of sugar secreted in twenty-four hours often reaches 0.5 to 1 pound (gm. 200 to 500). Of course the amount varies greatly according to the diet, mode of life, and treatment of the patient. The greatest amount ever known to be produced in twenty-four hours was more than 2½ pounds (gm. 1,000). The percentage of sugar in the urine varies between one half to one per cent at the lowest extreme, and eight to ten per cent as the maximum; usually it is about two to four per cent.

The most important tests for sugar in the urine are: First, Trommer's test: To urine in a test-tube sufficient potassium or sodium hydrate is added (say 1 part to 3 or 4 of urine) to make the reaction strongly alkaline; then a solution of sulphate of copper (about 1 part of the salt to 10 of water) is added, drop by drop. If the urine contain sugar, the hydrated cupric oxid, which is at first formed, is largely dissolved, and usually the fluid assumes a beautiful deep blue color. We ought, properly, to go on adding the sulphate of copper until the hydrated cupric oxid ceases to be dissolved. The urine is then heated, whereupon the cupric oxid is reduced and a yellow, or reddish-yellow, precipitate of cuprous oxid, or hydrated cuprous oxid, is formed. The application of heat should not be continued long after the precipitation begins to take place, lest the test be obscured; the reduction will go on even without heat. If the urine contains more than one half to one per cent, this test is perfectly reliable. If the reaction be a doubtful one—that is, if there be no precipitate of cuprous oxid, although the urine becomes yellow; or if cuprous oxid be finally precipitated when the urine cools—we should be cautious in making a diagnosis, as the urine may contain substances other than sugar capable of reducing the copper, such as uric acid, kreatinin, mucin, etc. Second, the bismuth test (Böttger's): Sodium hydrate or sodium carbonate is added to the urine, and then a small pinch of subnitrate of bismuth. Upon boiling, the urine, if it contains sugar, quickly assumes a perfectly black color, the oxid being reduced to bismuth or bismuth oxydul. It is simpler, and consequently universally customary at present, to make the bismuth test by adding to the urine about one tenth of its volume of the following solution (Nylander): 2 parts subnitrate of bismuth, 4 parts Rochelle salts, 100 parts of an eight-per-cent solution of sodium hydrate. After one or two minutes' boiling the fluid turns black. Errors with this test are most apt to occur if the patient has taken certain drugs, such as antipyrin, salicylic acid, or rhubarb. Third, the potassium test (Moore's): Potassium hydrate is added to the urine in the test-tube and the uppermost layer cautiously heated; if it contains sugar, the urine quickly assumes a deep brown color, as a result of the action of the potassium on the sugar, and this upper dark-colored layer contrasts strongly with the clear urine below.

[Fehling's test is justly a favorite in this country, and has the advantage of being applicable to the quantitative as well as to the qualitative analysis. The difficulty of its not keeping well can be met by having separate bottles for the copper and tartrate solutions, and making the mixture at the time of using the test. The Fehling's-test pellets put up by chemists are convenient for the qualitative analysis, but, on the whole, they are inferior to the solution.]

If the above-described tests leave us still in doubt, there can be only a small amount of sugar, if any, present. We may, however, attain certainty by employing the fermentation test (which causes a decomposition of the sugar into alcohol and carbonic dioxid), or circumpolarization (deflection of the plane of polarization to the right by the grape sugar). Certainly in no doubtful case should we omit to perform the fermentation test, for it is easy and gives absolutely unambiguous results. Further particulars in regard to these and in regard to the practical and likewise important phenylhydrazin test of E. Fischer (demonstration of osazone crystals), and also in regard to the quantitative estimation of sugar, may be found in works on medical chemistry.

Diabetic urine sometimes contains other varieties of sugar in small amounts—namely, levulose, which deflects the plane of polarization toward the left, and pentose; these are, however, of no practical importance.

The amount of urea is usually high. In the main, this is doubtless connected with the increased ingestion of albumen, but in severe cases it may be that the carbohydrates are so imperfectly appropriated that the albumen of the body is broken up and consequently contributes to an increase in the excretion of nitrogen.

Whether or not, under certain circumstances, especially in severe cases, and during diabetic coma, an abnormal increase of the albumen catabolism (so-called toxogenous albumen disintegration) also occurs, is not positively determined; von Noorden and others, however, think this probable. Uric acid is usually excreted in approximately normal amounts. The amount of phosphoric acid and sulphuric acid usually corresponds to the amount of urea, or, in other words, to the decomposition of albuminoids. The amount of sodium chlorid excreted depends, as in health, merely upon the amount ingested.

Hallervorden has discovered an important fact in regard to the excretion of ammonia. In many cases of diabetes, although not in all, it is much increased: 45 to 90 gr. (gm. 3 to 6), or even more, may be excreted in twenty-four hours. Despite this, diabetic urine has an acid reaction; and, as Stadelmann has shown, the basic elements are considerably out of proportion to the acids known to us. It is, therefore, evident that diabetic urine, since it contains a large amount of ammonium and yet has an acid reaction, must have among its constituents some unusual acid. The investigations of Minkowsky and Külz have shown that the acid is mainly oxybutyric acid, or, more accurately, left rotatory beta-oxybutyric, although sometimes still other acids are found in the urine, particularly volatile fatty acids and lactic acid (Rumpf). The oxybutyric acid is often excreted in large amounts, up to 2 or 3 ounces (gm. 50 to 100) daily. Together with this, two other substances usually appear in the urine, which are derivatives of the first—acetone and diacetic acid. The relative amounts of these three substances may, to be sure, greatly vary. Sometimes acetone alone is found in the urine; in other cases, only acetone and diacetic acid are found; while in a third type of cases all three "acetone bodies" appear in the urine. For a long time we were in the dark in regard to the development of the acetone bodies. We now know that they are not derived from the carbohydrates, nor, as was formerly supposed, from the proteid bodies, but from the fats (fatty acids). Strange to say, the acetone bodies appear in the urine only when the carbohydrate catabolism is limited. Where there is a simultaneous combustion of carbohydrates even an excessive fat diet does not produce any excretion of acetone bodies ("ketonuria"). During starvation, or in severe diseases, where the body uses up its own fat without the simultaneous administration of carbohydrates, acetone soon appears in the urine. The same thing occurs in severe diabetes, where only insufficient carbohydrate catabolism occurs, and more fat must therefore be burned up. Oxybutyric acid ($CH_3CHOHCH_2COOH$) is the first member of the series of acetone bodies; diacetic acid (CH_3COCH_2COOH) and acetone (CH_3COCH_3) are derived from it. The chemical demonstration of oxybutyric acid is possible only in a laboratory. The other two bodies, however, can be easily demonstrated, qualitatively.

Diacetic acid is the cause of the reaction which was first demonstrated by Gerhardt, which consists in the appearance of a Burgundy-red color on the addition of chlorid of iron. Acetone is shown by Legal's test: a few drops of fresh solution of nitro-prusside of sodium are mixed with the urine, and then sodic hydrate is added. The urine assumes a reddish color, which becomes purple-red or violet-red on the addition of acetic acid. If there is no acetone in the urine, this purple color does not appear on the addition of acetic acid. The urine then becomes yellow. The important relations of oxybutyric acid and the other acetone bodies to diabetic coma will be considered later.

The not infrequent occurrence of albumen in diabetic urine will be mentioned when considering the renal complications.

2. METABOLISM IN DIABETES. SOURCES OF THE SUGAR AND VARIATIONS IN ITS AMOUNT OCCASIONED BY EXTERNAL INFLUENCES.—Inasmuch as the presence of sugar in the urine is the most prominent symptom in diabetes, the question of its origin is all-important. One fact is indubitable—namely, that the secretion of sugar depends in large part upon the amount of potential sugar ingested—that is, upon the proportion of starches in the food. The amount of sugar excreted with the urine increases and diminishes with the amount of starchy food eaten. If a diabetic patient abstains totally for any length of time from such articles of food as contain starch, sugar will, in many instances, entirely disappear from the urine. In other words, the system of a diabetic patient is almost, if not quite, incapable of oxidizing sugar into carbonic-dioxid gas and water. In consequence thereof, the blood of a diabetic contains an abnormally large amount of sugar (0.2 to 0.5 per cent).

Although in a diabetic patient the absorption of oxygen from the atmosphere goes on as usual, yet there always remains a deficit in the excretion of carbonic-dioxid gas and water, corresponding to that portion of the ingested carbohydrates which is not oxidized.

The oxidation of sugar is not absolutely *nil* in diabetes. Külz has proved by numerous experiments that not all the starch is excreted in the form of sugar. In severe cases of diabetes the largest part of the ingested sugar is excreted as such, unoxidized, with the urine, but in mild cases only a small portion is thus excreted. It is, therefore, necessary to determine the degree of tolerance for carbohydrates in every individual case of diabetes; even in the same patient this tolerance is not the same at all times. Another very interesting fact was demonstrated by Külz. The diabetic patient has lost only the power of oxidizing that kind of sugar which turns the plane of polarization toward the right. The sugar which turns it toward the left (levulose), inulin, and also certain other related substances, such as mannite and inosite, are broken up even in diabetic patients in very large part, if not completely, so that the amount of sugar excreted in the urine is but little, if any, increased by the ingestion of these substances. The different cases vary in their relation toward milk sugars.

Yet the carbohydrates are not the only source of the sugar excreted by diabetic patients. In many cases, although not in all, sugar persists in the urine even when the patient receives a dietary absolutely free from carbohydrates. In such instances there can be no doubt that the glycogen, or the sugar which is formed out of it, is referable to the albumen of the food. In general, we may say that it is always a sign of a severe variety of diabetes to have a part of the sugar which is formed out of albumen, when carbohydrates are absolutely excluded from the diet, excreted unoxidized; while it is a proof that the case is a milder one to have the sugar vanish from the urine when the patient is put upon a diet which contains little, if any, carbohydrates.

Finally, regarding the question of the formation of sugar from fat, the opinions of the various investigators are still divided. It is certain that even with the most liberal fat diet an increase in the excretion of sugar never occurs in diabetes. In severe cases of diabetes, however, which have been put on an absolutely carbohydrate-free diet, sugar is occasionally excreted in such abundance that the proteids would not suffice as the sole source thereof. Nothing remains, then, but to assume a formation of the sugar from fat (Seegen v. Noorden, Pflüger). Muscular exertion is one of the other external influences which, aside from the nature of the nourishment, modify the excretion of sugar in diabetes. Muscular activity consumes mainly non-nitrogenous substances (i. e., the carbohydrates), and accordingly we find in diabetes that an increase of muscular exertion, other things being equal, diminishes the amount of sugar excreted. There are, however, interesting exceptions to this rule (Külz, v. Noorden)—i. e., cases where increased muscular exertion, with the diet remaining the same, does not reduce the amount of sugar excreted, and where it would therefore appear that the muscles are no longer able to work at the expense of the carbohydrates.

Emotional excitement seems frequently to increase the amount of sugar excreted. Increased external temperature (warm climate), on the other hand, is supposed to reduce the sugar excretion.

Intercurrent acute febrile diseases may cause a great diminution in the amount of sugar, but sometimes there is no essential change. Probably the altered diet of the patient plays an important part in this connection, although the modifications of metabolism occasioned by the high temperature or by the disease itself also exert some influence. Just before death the urine may become entirely free of sugar.

3. Constitutional Symptoms in Diabetes Mellitus.—In many of the milder cases there is for a long time little apparent disturbance of the general health. The patient is well nourished, and suffers little discomfort, except that he is rather easily fatigued, and somewhat inconvenienced by the polyuria and the polydipsia.

There are some diabetics who, for a long time, even with a decided excretion of sugar, feel subjectively almost entirely well. In the majority of the severer cases the system is deeply affected by the drain upon it. The patient becomes emaciated, weak, and easily exhausted, and at length there may be profound marasmus. Mentally the patient is apt to be depressed and irritable. The intellectual powers are not impaired, but there is indisposition to mental effort. The temperature is normal or subnormal. Fever invariably indicates some complication.

4. Symptoms referable to the Digestive Organs.—We have already mentioned the excessive thirst experienced in diabetes. This may be a source of great discomfort, obliging the patient to drink at short intervals, even through the night. The interdependence of polyuria and polydipsia is not yet fully understood. The most natural view seems to be that the increased excretion of water by the kidneys is the primary factor, and the increased thirst secondary thereto. One cause of the polyuria is the excretion of sugar, in order to dissolve which a large amount of water is necessary, but certain factors would also seem to be implicated. That they exert some influence is rendered probable by the fact that the amount of urine does not always correspond to the amount of sugar excreted. A very large amount of urine may be excreted containing little or no sugar; and, on the other hand, there are genuine cases of diabetes mellitus where the amount of urine is normal, and the patient feels no unusual thirst (*diabetes decipiens*). It has also been suggested that the sugar may irritate the nerves of the mouth and throat, and thus cause thirst. According to this idea, the polyuria would be partly the result of the excessive ingestion of liquids. The abnormally great appetite in diabetes seems to be due to defective assimilation of the food. Many patients are never able to eat enough. They have a longing, in many cases, for carbohydrates. Occasionally the hunger becomes ravenous, and is associated with headache and a general sense of weakness, all these symptoms being alleviated when food is taken. This rule has rare exceptions in which the appetite is not unusually great, even though the case be severe.

The tongue is frequently dry; it is broad and thick, with an irregular and fissured surface, sometimes coated and sometimes red. The gums may be spongy, and may exhibit a tendency to bleed. A very characteristic condition is necrosis along the free borders of the gums, probably due to an enfeeblement of the resisting power of the tissues. This is particularly apt to lead to a gradual loosening of the incisors, so that at last they drop out. Many a case of diabetes has first been recognized by this condition of the teeth.

The teeth frequently decay rapidly. The saliva is invariably found to give an acid reaction. This is true of the isolated secretion of the parotid gland also, and is said to be due to the presence of lactic acid. It is only in exceptional instances that sugar can be demonstrated in the saliva. In severe cases thrush sometimes forms upon the soft palate.

Special gastric symptoms are usually absent. There may be a dilatation of the stomach, due to the great quantity of food ingested, but this has no practical importance. If disease of the stomach does develop (e. g., chronic gastritis) the complication is an unfavorable one. There is usually constipation, but sometimes there is a severe, though temporary, diarrhea. An increased fat content of the stools points to disease of the pancreas (*vide infra*). The liver and spleen are seldom much affected; the liver rarely is somewhat enlarged. Jaundice is frequently observed, but it is always referable to some complication. As a rule, the secretion of the bile goes on as in health.

5. SYMPTOMS REFERABLE TO THE RESPIRATORY ORGANS.—In many cases the organs of respiration are unimpaired for a long while. It may be mentioned that often patients have a decidedly fruity odor to the breath (acetone odor, due to the excretion of acetone by the lungs). In the later stages of the disease pulmonary complications are very frequent. First in importance is tuberculosis of the lungs. This is particularly apt to occur in the severe forms of diabetes in youth, and it may pursue a rapid and fatal course. The diagnosis can be settled by the discovery of tubercle bacilli in the sputum. Next in point of frequency is pulmonary gangrene. Sometimes there is a diffuse gangrenous process, and sometimes there are isolated foci of necrosis, which become liquid, and have an acid reaction, but they often have comparatively little odor. The expectoration in these cases may be odorless. Croupous pneumonia may also occur. It often terminates unfavorably, and may, as we have ourselves observed, result in gangrene.

6. SYMPTOMS REFERABLE TO THE CIRCULATORY SYSTEM.—In many instances the circulatory apparatus presents no special lesions. The pulse is either of a normal rate or a trifle slow. It is usually soft, although exceptionally it may exhibit increased tension. There is often distinct evidence of cardiac weakness, the pulse is small, intermittent, sometimes very slow (50 or even 40 beats per minute), and sometimes accelerated (100 to 120 beats per minute). There are shortness of breath, faintness, nausea, and the like. Sometimes sudden and profound cardiac disturbance occurs, and this may occasion speedy death (*vide infra,* coma). In all these cases, however, this is usually not a result of the diabetes, but a coexistent phenomenon. The cardiac disturbance occurs particularly in elderly obese diabetics, and depends upon a simultaneous coronary and aortic sclerosis, upon alcoholic heart disease, etc.

7. GENITO-URINARY SYMPTOMS.—Despite the great demands made upon them, the kidneys often maintain a normal condition. As we shall see when we come to the pathological anatomy of diabetes, the kidneys are often very large. Sometimes a chronic nephritis is developed as a complication, usually in the later stages of the disease. The urine contains albumen, and there are œdema and other symptoms of renal disorder. The cause of albuminuria in diabetes does not seem to be the same in every case; sometimes it is due to changes in the kidneys resulting from the persistent excretion of abnormal urinary constituents, which have an injurious effect upon the renal epithelium.

In this connection we must think partly of the sugar, and also perhaps of the other substances, such as acetone, diacetic acid, and oxybutyric acid, etc. In the majority of cases, diabetes and nephritis seem to be simultaneous and coördinate results of the same cause, such as arteriosclerosis, alcoholism, gout, etc. This follows already from the fact that kidney complications are much more frequent in the diabetes of elderly and obese individuals than in the severe forms that occur in youth. Finally, it must not be forgotten that other complications of diabetes—e. g., pulmonary tuberculosis—may promote the development of nephritis. It is an interesting fact that in diabetes, glycosuria and albuminuria may, to a certain extent, alternate with each other. If the amount of albumen in the urine becomes considerable, the amount of sugar in the urine usually undergoes marked diminution, or disappears entirely. Severe suppurative pyelonephritis also occasionally occurs in diabetes. Cystitis may likewise develop, and may be associated with fermentation of the urine in the bladder and pneumaturia.

Saccharine urine is apt, as it decomposes, to cause irritation of the skin. This is the explanation of the troublesome pruritus pudendi, which is especially marked in women. It may, indeed, be this symptom which first directs attention to the disease. Sometimes the external genitals are attacked by eczema or furunculosis. Men often suffer from balanitis, with inflammatory phimosis, or paraphimosis. A frequent and important symptom in men is impotence. This sometimes occurs very early in the disease, but it may afterwards undergo improvement. The origin of it is probably referable to degenerative changes in the nervous system (*vide infra*). Some authorities state that diabetes is apt to occasion atrophy of the testicles.

8. Disturbances of the Organs of Special Sense.—An important and not infrequent result of diabetes is cataract. This may occasion almost total blindness. The cause of cataract in diabetes is not known. It was formerly supposed that the sugar in the blood absorbed water from the crystalline lens, and thus occasioned its opacity; but this has not been confirmed. Diabetic patients are subject to disturbances of accommodation. Paralyses of the larger eye muscles have also been observed. Retinitis and atrophy of the optic nerve may occur, but they are very rare. Purulent choroiditis, if it develops, is probably merely a chance complication.

None of the other special senses is peculiarly affected in diabetes.

9. Cutaneous Affections.—In most cases the skin is remarkably dry and rough. There may, however, be abundant perspiration. Several authorities claim to have found sugar in the perspiration, but this statement has not been confirmed by later investigators. Sometimes there is a troublesome pruritus. We often see a rapid loss of hair and a shedding of the nails. In many cases there is great tendency to furunculosis. This may be the first symptom to suggest the existence of diabetes. In the later stages there are sometimes extensive carbuncles, and multiple phlegmonous abscesses in the subcutaneous cellular tissue, which may prove fatal. Probably they are complications, like the frequent occurrence of pulmonary tuberculosis in diabetes. Occasionally we have seen a pemphigus-like eruption on the skin. This, in one case, developed into a localized gangrene. Gangrenous processes have been repeatedly observed, in particular necrosis of one or more toes, which may take the form of perforating ulcer (*mal perforant*), or rarely gangrene of an

entire extremity. This gangrene usually is due to arterial sclerosis; in some cases its cause is obscure. The gangrene of the toes may be apparently the first symptom of the disease, and it may be very slight. It is not until all therapeutic and operative procedures prove unavailing that suspicion is aroused, leading to the discovery of diabetes. We have often seen cases of this sort, which usually turn out badly, in obese and elderly individuals.

Œdema of the subcutaneous cellular tissue may occur independently of nephritis. It is then probably occasioned by the cardiac weakness.

10. Symptoms referable to the Nervous System.—It has already been mentioned that there is frequently in diabetes a moderate disturbance of the whole nervous system, as indicated by headache, physical and mental hebetude, and depression of spirits. There are, besides, a number of nervous complications which are probably referable to the irritative or degenerative effects upon certain nerve areas of the abnormal products of metabolism formed in diabetes. This is the explanation of the comparatively frequent appearance of neuralgia, most often in the form of sciatica. This is probably due to neuritis of the nerve. Bilateral sciatica may be one of the first symptoms of the disease, and a very obstinate one. We also meet with occipital neuralgia, facial neuralgia, and pain resembling hemicrania. Sometimes, also, we observe localized anæsthesia of the skin and peripheral palsies. These are probably due to degenerative changes in the peripheral nerves. We have ourselves seen, in one case, paralysis of the peroneal nerve, due to peripheral neuritis. Another symptom which should be grouped with those just considered, is one which was first pointed out by Bouchardat—viz., the comparatively frequent abolition of the patellar reflex in diabetes. It should be said, however, that we have repeatedly seen cases, of the most severe form, in which the knee jerk remained perfectly normal. If the reader will imagine a case in which absence of the knee jerk is associated with neuritic pains in the leg, he will understand the reason for the term "diabetic pseudo-tabes." The pathological basis of the symptoms just enumerated has been repeatedly shown to be neuritic degeneration. In some few cases there may even be slight changes in the posterior columns of the spinal cord.

The most important nervous symptom of all is a peculiar disturbance which occurs in a considerable proportion of all cases with more or less suddenness, and usually terminates in a surprisingly speedy death. This strange phenomenon was first thoroughly investigated by Kussmaul, although known long before. It is termed diabetic coma. v. Noorden observed 169 deaths in coma in 292 fatal cases, an amount equal to fifty-eight per cent.

Diabetic coma sometimes develops without any evident cause. In other instances it is apparently brought on by violent muscular exertion, mental excitement, or some trifling illness, such as gastric catarrh, bronchitis, or sore throat. Frequently certain mild prodromata herald its onset. There may be nausea, headache, pains in the stomach or back, a sense of thoracic oppression, and general uneasiness. Soon the picture changes. The patient becomes peculiarly apathetic, somnolent, and, finally, complete loss of consciousness supervenes (coma). Only exceptionally does a stage of marked excitement precede the development of coma. Convulsions occur only very rarely in true diabetic coma. On the other hand, a peculiar alteration in respiration (dyspnœic coma) is very common and characteristic. The breathing becomes remarkably deep and noisy. Its rate may remain nearly normal, or it may be considerably increased, so as to justify the term "diabetic dyspnœa." The patient is not cyanotic. The pulse is usually very rapid and small. The temperature gradually sinks, and has in repeated instances fallen to 86° F. (30° C.), or even lower. P. Krause has found in our clinic an unusually striking diminution in the intraocular tension (*hypotonia bulbi*). This symptom seems to be almost constant. An acceptable explanation of it cannot as yet be given. On ophthalmoscopic examination, Krause once found a remarkable white border on the edges of the retinal vessels. This white border is connected with the increased fat content of the blood. The blood, on venesection, showed a very pronounced lipæmia. Lipæmia has been repeatedly observed in severe cases of diabetes, and especially in coma. The cause of this remarkable phenomenon is also not entirely explained. Primarily, we must think of an absence, or, at least, an inefficiency of the lipolytic ferment (the so-called lipose) in the blood. In most instances, also, the breath has a very noticeable odor, resembling fruit or chloroform, which may be perceived on entering the room. Even the urine may have this same odor, and it almost invariably becomes dark red on the addition of ferric chlorid, and gives a distinct acetone reaction (*vide supra*).

True diabetic coma must be differentiated from the other infrequent comatose conditions, such as uræmia, coma in hepatic cirrhosis, and apoplexy. True diabetic coma ends, after it is once fully developed, almost without exception in death. It usually lasts one to two days. Only occasionally do we see cases where a beginning coma disappears.

As regards the cause of the coma, the above-mentioned investigations of Stadelmann, Minkowski, and others have thrown some light upon the subject. With the greatest probability we can regard diabetic coma as an acid intoxication. We have already learned of the occurrence of large quantities of oxybutyric acid in diabetes. During coma Magnus-Levy found an excretion in the urine of 3.5 to 5.3 ounces (gm. 100 to 160) in twenty-four hours, and has also found large quantities in the organs after death. These amounts are amply sufficient to produce an acid intoxication. The various alkalies of the blood are neutralized by the acid, so that the CO_2 can no longer enter into combination, and therefore cannot be discharged from the body tissues. This CO_2 congestion in the tissues, particularly in the brain, produces the phenomenon of coma. In animals similar conditions have also been produced experimentally with other acids. There is a corresponding marked decrease in the CO_2 content of the blood in diabetic coma.

Pathological Anatomy and Histochemistry of Diabetes Mellitus.—If we exclude the organic diseases, such as pulmonary tuberculosis and nephritis, which are merely complications, the pathological changes in diabetes are trifling. Bernard's discovery, that an injury inflicted in a certain spot on the floor of the fourth ventricle produces glycosuria in animals, has directed the attention of investigators to the condition of the nervous system in this disease. In some instances, tumors, sclerosis, or similar troubles have been found in the medulla and cerebellum; but in these cases there was evidently a symptomatic glycosuria and not an idiopathic diabetes (*vide supra*). In idiopathic cases the central nervous system presents no striking macroscopic changes. By means of the microscope, Frerichs has found lesions of the medulla oblongata in frequent instances. The minute blood vessels are widely dilated; there are small capillary hemorrhages, some of a more recent and others of a more remote date, and occasionally there are microscopic foci of myelitis. The nervous elements proper, the nerve fibers and ganglion cells, betray no alteration; and, as the changes enumerated are not invariably present, their significance is dubious.

The stomach and intestines present no constant alterations of importance.

The liver has naturally been the object of repeated and careful examinations because of its well-known part in the manufacture of glycogen. Yet this organ seldom presents any special abnormality. It is usually of the natural size, and it may contain either considerable or very little blood. The amount of glycogen in the hepatic cells can be demonstrated with iodin by a microchemical reaction. It appears that, other things being equal, there is less glycogen present than normal. It is usually found only in the cells situated upon the periphery of the lobules, and in small quantities. In an extremely advanced case of diabetes Ehrlich obtained, by means of a hollow needle, small amounts of the hepatic parenchyma for examination during life, and found that glycogen was almost completely absent. In other cases the liver has been examined as early as possible after death, and, with rare exceptions, has presented no trace of glycogen.

The spleen is usually of normal size. Occasionally it is atrophied, or, on the other hand, slightly enlarged. No other changes in it have been observed.

Not a few cases have presented a striking atrophy of the pancreas (Bouchardat), a fact which has acquired great significance from Minkowsky's discovery, already mentioned, that glycosuria may come on after extirpation of the pancreas. It is uncertain whether the cœliac plexus may also sometimes share in the atrophy of the pancreas. We must also state, however, that atrophy of the pancreas is not at all a constant lesion in diabetes, so that it is not proper to regard the disease of the pancreas as the special cause of diabetes in all cases. Lately in repeated instances the pancreas has been found perfectly normal, even upon the most careful microscopic examination. In the diabetes of elderly individuals with arteriosclerosis, it is possible that the arteriosclerosis of the pancreatic artery and the consequent disturbances of that organ play a rôle.

The kidneys are usually enlarged, from functional hypertrophy. Ehrlich discovered in them a glycogenic degeneration of the loops of Henle. The epithelial cells in the loops are enlarged, and the protoplasm in these cells, although apparently homogeneous, is found, by the addition of a solution of iodin in mucilage, to contain glycogen in flakes and clumps of varying size. How important this glycogenic degeneration of the kidneys may be has not yet been determined. Perhaps the glycogen represents sugar which has been absorbed by the cells. That chronic nephritis may complicate diabetes has already been mentioned. We have already referred to the increased sugar content of the blood. It is usually somewhere between 0.2 and 0.45 per cent of sugar, while in health the blood rarely contains over 0.1 per cent. An interesting fact, discovered by Williamson and by Bremer, that the blood of diabetics does not, like normal blood, stain blue with alkaline methylene-blue solution, but only a pale green, seems to depend upon this abnormal sugar content of the blood, or possibly also upon other chemical changes (acidosis?).

The lymph, and such serous effusions as are found, contain sugar, but the saliva, perspiration, bile, gastric juice, and other secretions rarely furnish evidence of its existence.

Varieties, Course, and Prognosis of the Disease.—The study of a large number of cases of diabetes will show great variations in the course and duration of the disease. As already stated, we may in practice distinguish between mild and severe forms of diabetes. Following Seegen's suggestion, this division is usually based upon the relation between the sugar excreted and the character and amount of the food eaten. Those cases are regarded as belonging to the mild form in which the sugar disappears from the urine when the patient takes food containing no carbohydrates. Some patients with this milder form may ingest small amounts of the amylacea, particularly if they take a proper amount of physical exercise (*vide infra*), without bringing on glycosuria. In the severe form of diabetes, on the other hand, the excretion of sugar in the urine persists even on a purely meat diet, and if any carbohydrates are taken, there will be a corresponding and large increase in the amount of sugar in the urine, as soon as half an hour to an hour thereafter.

In some cases the sugar content disappears only when carbohydrates are withdrawn from the diet for a long time. These have been designated as cases of "medium severity" by Naunyn. There are altogether between the mild and severe cases all kinds of transitional types. Of just as much practical importance as the excretion of sugar are the clinical signs which sometimes indicate, at the first glance, that an individual suffers from the mild or the severe form, as the case may be. The severe cases usually attack youthful individuals; the patient loses flesh rapidly, he is prematurely aged, his face becomes narrow, and is apt to assume a peculiar expression of quiet melancholy; he is languid and feeble, and the urine presents all the indications of diabetes in an extreme degree. The milder cases, on the other hand, are more apt to occur in later life, the patient has only moderate discomfort, and usually retains considerable strength and energy; he is but little, if at all, emaciated, and even the changes in the urine are so slight that they are not recognized until the attention is especially directed to them. It must be noted, however, that an apparently mild case of diabetes may become severe. Sometimes, also, the behavior of the case as to the excretion of sugar remains like that of the mild variety, and still, fatal complications occur at last, such as coma, necrotic processes, and pulmonary tuberculosis.

In general, the clinical course of diabetes presents great variations. In a few instances the disease occupies only a few weeks, and it may almost be termed "acute diabetes." These cases with absolutely unfavorable prognosis are especially observed in children and youthful individuals somewhere between the ages of twelve and eighteen years. We have seen quite a number of such cases that began almost suddenly, without demonstrable cause, and usually after a few months, or even weeks, ended fatally—generally in diabetic coma. The majority of cases of severe diabetes last about two or three years, while the milder forms may last ten to twenty years, or even longer. The patient's condition may vary from time to time. We have repeatedly seen the sugar disappear temporarily from the urine, and the patient apparently completely recover, but sooner or later the disease breaks out again. These cases, which are commoner in old people, have been called "intermittent diabetes."

The relapse is often brought about by emotional excitement or some grave error in diet. Again, the disease may become apparently stationary and the patient enjoy comparative comfort for years.

Different cases also differ in the relative severity of particular symptoms. Thus, the clinical picture presented by diabetes may be modified by the general constitution of the patient—for instance, his corpulence or emaciation; by such complications as diseases of the lungs, kidneys, or brain, or syphilis and gout, and by many other conditions. In practice, especially in practice among the better classes, it is very important to have a knowledge of the milder forms of diabetes, in which sugar is present in the urine only at times, and not in very large amount. The more we are accustomed to examine every urine for sugar, the oftener we find such cases, as to the significance of which our knowledge is still very slight. The glycosuria of the corpulent is worthy of special consideration. It is, as we have said, particularly apt to occur in obese beer drinkers, and it is not uncommon in patients who have formerly suffered from true gout. The tendency to the formation of furuncles and cataract is noteworthy. We must also mention the cases of diabetes in neurasthenics—that is, cases where there is sugar in the urine, and where the chief complaint is of general nervous symptoms, pressure in the head, anxiety, a dull feeling, incapacity for mental work, neuralgic pains, etc. Finally, we would point out an especial clinical form of diabetes, in which glycosuria occurs in patients with severe general arteriosclerosis; usually the patients are men in middle or advanced life. Not infrequently the condition seems referable to long-continued mental excitement or exertion. The amount of sugar in the urine is not large, and the sugar usually entirely disappears with appropriate diet; still, this form cannot be unqualifiedly regarded as mild, for we must bear in mind, also, the serious results of arteriosclerosis. There are particularly apt to be cardiac symptoms referable to arteriosclerosis of the coronary arteries. In all these varieties of diabetes there may be no polyuria or thirst, so that an error in diagnosis is easy unless a careful examination of the urine is made.

We should also mention the interesting fact, of which Frerichs reports some striking examples, that diabetes mellitus in a few exceptional cases may gradually change into diabetes insipidus (see the following chapter). The reverse change is also said to occur. We ourselves have never seen such a transition.

The usual termination of diabetes is death. We have already seen how great a difference there may be in the length of time preceding the fatal termination, and in what various ways it may be brought about. The most frequent immediate causes of death are marasmus, diabetic coma, pulmonary consumption, nephritis, furunculosis, or the development of carbuncles.

The question of the curability of diabetes is difficult to answer. A number of observations seem to speak for the occurrence of a complete cure, but this is rare, and is possible only in the milder cases. It should also be borne in mind that, as we have said, apparent recovery does not exclude the possibility of a fresh outbreak of the disease.

Theoretical Discussion of the Nature of Diabetes.—The essential fact which demands explanation is the excess of sugar in the blood. The blood of a healthy man does not contain over 0.10 per cent of sugar, while that of a diabetic has 0.22 to 0.44 per cent. The source of this sugar is probably the same as of the sugar normally contained in the blood. The largest part of the sugar probably comes from the carbohydrates ingested as food. These are changed into grape sugar partly by the action of the saliva, but mainly by the pancreatic diastase. The grape sugar is absorbed as such by the portal tributaries, and carried to the liver. Here a large part is stored up as glycogen, and a small part is deposited in the muscles likewise as glycogen. The liver is the main reservoir for glycogen. It gives this off to the muscles in the form of sugar when the latter require it for their work. The sugar probably enters the muscle cell as glycogen, and is used up as such during the muscle work. Glycogen is not derived exclusively from the carbohydrates, but may also come from albumen. This has been proved by numerous experiments, and clinical experiences with the severe forms of diabetes confirm it. The formation of sugar from proteid is also proved by the experiment of Mehring, in which he fed phloridzin to fasting animals that had no more glycogen stored up in their bodies. A diabetes developed, and the excreted sugar could only have been derived from the disintegrated body proteid. The formation of glycogen or sugar from fat has not been positively proved, but is, however, assumed by many investigators (*vide supra*).

It would seem, therefore, that the sources of sugar are the same in diabetes as in health. We have next to seek for the reason of its excessive accumulation in the blood. Under normal circumstances the sugar present in that fluid rapidly undergoes decomposition into other substances. In health there is no great excess of sugar in the blood, even upon a diet extremely rich in starch, and it is possible to eat large amounts of sugar (*vide supra*, page 93) without any glycosuria. It is certain that the diabetic has, either entirely or in part, lost the power to oxidize carbohydrates. This is demonstrated with certainty by the determination of the so-called respiratory quotient—i. e., the relation of the amount of CO_2 excreted to that of oxygen absorbed. The administration of carbohydrates does not raise the respiratory quotient in diabetes, as it always does under normal conditions. As the sugar is not further oxidized, it accumulates in the blood (glycæmia), and is excreted as useless by the kidney. The question now arises: Upon what does this inability to destroy the sugar depend? It has been thought that there is lacking in diabetes a particular "glycolytic ferment," which is perhaps furnished to the blood by the pancreas, and stimulates the decomposition of sugar. But all attempts to demonstrate such glycolytic ferment and its absence in diabetes have failed up to now. It must also be considered that the main destruction of sugar does not take place in the blood, but in the tissues, particularly in the muscles. As already mentioned, the sugar is deposited in the tissues as glycogen. Now, in diabetes, there is always a striking deficiency of glycogen in those tissues (the liver and muscles) which, under normal conditions, form glycogen and store it up. We might therefore explain the glycæmia of diabetics by assuming that their organs have lost their ability to transpose sugar into glycogen, and to retain a part, as such, in their cells, and to destroy the remainder as needed (v. Noorden). In this case, too, the action of a ferment coming normally from the pancreas may be lacking.

As we see, the riddle of diabetes is still far from solution. But the questions to be solved are being constantly narrowed down more and more, and the direction from which it is to be assumed the solution will come (ferment action, internal secretion) is gradually becoming more apparent.

Diagnosis.—For the diagnosis of diabetes mellitus it is indispensable that sugar should be demonstrated in the urine. We have also to decide whether the condition be a temporary or a permanent one, or, in other words, whether we have to deal with mere glycosuria (*vide supra*) or genuine diabetes mellitus. This point is to be determined by means of the symptoms and general course of the disease.

Diabetes often exists for some time unsuspected even by the physician. It may therefore be well to name over the symptoms which may be the first to attract the patient's attention, and which should therefore always suggest to a physician the possibility of the existence of diabetes. They are (1) languor and debility; (2) furunculosis; (3) pruritus pudendi in women, balanitis in men; (4) cataract; (5) sciatica, especially if bilateral; (6) impotence. We should be in the habit of examining the urine for sugar in all cases when complaint is made of indefinite symptoms, which cannot be explained without further evidence, especially if the patient is fat or nervous, and also if he is elderly and shows signs of arteriosclerosis. We should furthermore never limit ourselves to the examination of a single specimen of urine.

If symptoms similar to those just enumerated lead to an examination of the urine, and the result of this is ambiguous, it is advisable to have the patient partake of a meal rich in starchy elements, and to examine the urine thereafter, preferably by means of the fermentation test. If even then no sugar is found, diabetes does not exist.

Treatment.—Medical science does not possess the power to cure the disease, but it can greatly benefit the patient both by alleviating his symptoms and by shielding him, at least for the time, from many of the secondary effects.

The therapy of diabetes must, in the first place, be a dietetic one. We will have to offer the body not only a substitute for the loss of sugar combustion with its consequent diminution in energy, but also to reduce the abnormally high sugar content of the blood and the glycosuria. For the pronounced glycæmia is certainly not without effect upon the tissues, and the presence of sugar in the urine may also lead to undesirable sequelæ, such as pruritus and balanitis. We must warn against the one-sided conclusion that we accomplish our whole duty by reducing the amount of sugar contained in the urine to a minimum. The general condition of the patient should invariably be considered. There can be no doubt that a diabetic patient whose strength is well maintained is better off, even if there is considerable sugar in his urine, than one whose urine contains less sugar, but who yet is daily growing weaker.

The first problem that a physician has to solve in treating a diabetic is to find that diet with which, on the one hand, the glycosuria is reduced as much as possible, and with which, on the other hand, however, the patient feels at his best and his nutrition suffers the least. The problem must be met in each separate case according to the individual circumstances. For it is clear, for example, that an entirely different diet is suitable for the obese diabetic than for the emaciated one. Furthermore, the variety and degree of metabolic disturbance certainly vary in the different cases, so that each patient must really be the object of special study. Such an exact individual examination is hard to carry out in private practice. It is, therefore, advisable, if possible, to observe every diabetic, at least for a time, in a clinic or a good sanatorium, carefully to control the amount of food and the excretion of sugar, etc., and from this to prescribe the diet that must be kept.

The establishment of the dietary rules for diabetics is best begun by determining what tolerance for carbohydrates there may be in each particular case. The principle of dietetic treatment of diabetes does not consist in the simple withdrawal of all carbohydrates, but rather in allowing each patient as much of such substances as his body is in a condition to appropriate. This amount varies greatly in different cases, and also in the same patient at different times. We must first determine whether the case belongs to the severe or the mild type, as above defined—that is, whether sugar entirely disappears from the urine if no carbohydrates whatever are allowed. If it does—that is, if we are dealing with the milder form of diabetes—we then proceed, after the urine has been freed from sugar, to give gradually increasing amounts of carbohydrates, at the same time examining the urine every day in order to see whether the starch ingested is completely oxidized in the blood. The more carbohydrates that can be allowed without the appearance of sugar in the urine, the more favorable the case. When we have in this manner measured the ability of the system to utilize carbohydrates, the patient is kept for a considerable time upon such food as his system can manage completely to appropriate. The experience of physicians has shown that this sort of limitation of the carbohydrates may have a permanent beneficial influence, inasmuch as after the patient has adhered for a considerable time to a diet which keeps the urine free from sugar, the tolerance of the system for carbohydrates increases. We may, therefore, try gradually to make some increase in the amount of carbohydrates, and we shall not infrequently find that after persistence in such a diet for a considerable time, the patient will bear more carbohydrates without the return of glycosuria. Thus, guided by constant examinations of the urine, we can easily prescribe the correct diet for the individual patient.

If we are dealing with the severe form of diabetes—that is, if sugar is still excreted in the urine, even when the diet consists exclusively of meat and fat—we may try the effect of withdrawing all carbohydrates completely for a considerable time. Many physicians (Cantani, Naunyn, and others) have observed that an absolutely strict diet of this sort, excluding all carbohydrates, may have a very favorable therapeutic effect, so that finally the patient will regain a certain tolerance for carbohydrates; but it is precisely in these severe cases that, in our opinion, it is improper to be guided too much by general rules. We should consider not only the character of the urine, but the condition of the individual in other respects—his subjective sensations, his weight, and his strength. Many of these severe cases undoubtedly feel better when they are allowed a moderate amount of carbohydrates than when they are on a purely nitrogenous and fatty diet, despite the somewhat greater amount of sugar excreted. Particularly when the appearance of acetone, diacetic acid, butyric acid, and the like in the urine, suggests a considerable destruction of the albuminoids of the body, and perhaps an impending diabetic coma, practical experience has shown that we should, under no conditions, insist upon a too strictly nitrogenous diet, but should try, by the cautious administration of carbohydrates, to improve the general state and avert the danger of coma.

As we have already mentioned, it is decidedly advantageous to make up for the diminution in carbohydrates by giving other food which does not contain nitrogen. The most natural thing, certainly, is to allow an abundance of fat as a substitute for the carbohydrates, and practical experience is, as regards this matter, in entire unison with theory. Fat is well borne by most diabetics, and it may be allowed in the form of butter, cream, salad oil, and the like. It should be particularly recommended to emaciated patients, if they can digest it. Sometimes cod-liver oil may be prescribed for a time. It is just in the administration of fat to diabetics that we must carefully individualize. First of all, we must take great care that no digestive disturbances develop, and it is self-understood that in well-nourished, or even obese diabetics, a too liberal fat administration is partly unnecessary and partly harmful. In these cases we must try to bring about a reduction of the body fat. Anyway, an urgent warning must be sounded against unintelligent overfeeding in diabetes. This also applies to the administration of proteids. The profuse meat rations, which were formerly sometimes in vogue, are by no means advantageous for diabetic patients. The entire dietary for a diabetic should be so arranged that the excretion of sugar is kept down to the lowest possible point, without a simultaneous development of ketonuria. The patient at the same time should feel as well and as energetic as possible, and maintain his average body weight. As to liquids, water in the form of the alkaline mineral waters or simple tea may be permitted, according to the desires of the patient. Alcoholic drinks should, in general, be very much restricted. Alcohol does not come into consideration from the standpoint of tissue economy. Small amounts of wine may occasionally be permitted, but the use of large amounts, which might be considered solely for the purpose of saving the body tissues, is strongly to be deprecated, inasmuch as the toxic action of the alcohol upon the life of the cells is to be feared. Beer should be absolutely forbidden, likewise champagne.

With a view of aiding the physician in preparing a bill of fare for his individual cases, we append a list of the separate foods which are available for the nourishment of diabetic patients: 1. A diet which contains almost no carbohydrates would be as follows: On waking, tea or coffee with cream, but no sugar; for breakfast, eggs, ham, cold meat; at noon, broth, not thickened, fish with butter, meat with greens or salad; for supper, tea, eggs, cold meat, cheese; in addition the patient may drink water or a moderate amount of claret. The following articles are almost absolutely devoid of carbohydrates: all sorts of meat, ham, smoked meat, tongue, all sorts of fish, crabs, caviare, oysters, sardines, eggs, green-colored vegetables, lettuce, endives, cress, asparagus, cauliflower, spinach, cucumbers, red and white cabbage, string beans when young, sorrel. Of course, if these are boiled, only butter can be added, or meat, but no cereal. Other articles of this class are all sorts of cheese; such fatty foods as butter, olive oil, and cream; meat broths, artificial meat peptone, meat extract, somatose, mineral waters, and light claret. 2. Articles which contain a small amount of carbohydrates, and which, therefore, may be allowed in moderate amounts if there is a certain degree of tolerance for carbohydrates (*vide supra*): rusks, rye bread, milk, cocoa, turnips, kohl rabi, radishes, nuts, sour fruits—such as apples, currants, and cherries. 3. Articles which contain a large amount of carbohydrates, and are therefore to be prohibited or allowed sparingly, according to individual circumstances: all sweet dishes, cake, honey, potatoes, oatmeal, rice, peas, beans, lentils, sago, sweet fruits, sweet wines, and beer.

It is well worth noting that the diet list must not be constructed merely upon the basis of the amount of carbohydrates in the food substances. For, as recent experiences have frequently shown, the carbohydrates in the various food stuffs have not all the same action, and furthermore, there are often striking individual differences in the effect of the same carbohydrate upon various patients. Milk, particularly, is a very useful food for many diabetics, since milk sugar is obviously better utilized by many patients than grape sugar. This, however, is not true of all cases. Again, v. Noorden has called attention to the fact that oatmeal is very well borne by many diabetics. In the oatmeal treatment of v. Noorden, the patient receives 8 ounces (gm. 250) of oatmeal in the form of soup daily for three or four days, besides about 6.5 ounces (gm. 200) of butter, and 5 to 8 eggs or 3 ounces (gm. 100) of vegetable albumen (no meat!). With this diet, which may be alternated with some other strict dietary regimen, many diabetics lose their sugar with surprising rapidity. In other cases the oatmeal cure fails absolutely, and we are not yet able to say upon what these differences in result depend. Rice which has been cooked a long time (three hours) is also sometimes better borne than other carbohydrates. This also applies to certain fruits, such as apples, cherries, and strawberries. Briefly, we see how impossible it is to lay down dietary regulations which will apply to all diabetics. In general, treatment must always be begun with a very strict diet, and then, while still keeping the general condition and the state of the urine under consideration, some carbohydrates may be gradually permitted. In the milder cases it is often desirable to prescribe a strict diet for a period of one half to one week at regular intervals of about two or three weeks. Between these periods the patients may be permitted greater freedom. That, on the appearance of acetone and diacetic acid, the diet must not be too restricted, has already been mentioned.

Besides the diet, in the narrower sense, the application of general physical and dietetic remedies is of great importance. The proper regulation of the mode of life, the avoidance of mental excitement and overexertion are of importance. The degree of exercise must also be carefully prescribed. Strong, well-nourished diabetics may walk, undertake easy mountain climbs, do gymnastics, swim, row, etc. Thin, weak patients require rest (fresh-air rest cures). Certain external physical measures which stimulate metabolism may have the most beneficial effect. These are baths (salt, CO_2, and electric), rubbings, careful douches, massage, and, under certain circumstances, in strong patients, also careful sweat cures (electric-light baths). The constitutional effect of climate (air, sun, warmth) must also not be underestimated. Some patients profit very much from a trip to the mountains or seashore, or a winter's stay in the south. Finally, strict attention must be given to the toilet of the mouth, to prevent the teeth from becoming carious.

The hardest thing for diabetics to do without is bread. Every physician who has had much experience with diabetic patients can relate experiences of the cunning of patients in satisfying their unconquerable longing for this forbidden article by the employment of underhand means. Hence many attempts have been made to furnish special sorts of bread for diabetic patients poor in carbohydrates. Most of these preparations, however, the patients do not like. Chiefly to be recommended are gluten bread and aleuronat bread. Finally, it is well to employ a saccharin to sweeten food and drink, particularly tea and coffee, provided the saccharin does not produce dyspepsia and also proves agreeable to the patient.

Of internal remedies in general, no great influence upon the disease is to be expected. Nevertheless, occasional use of them may be made. Opium should be named first. One good effect of opium is that it lessens the annoying thirst. It sometimes also causes decided diminution in the amount of urine and sugar excreted. It is further indicated when there is general restlessness or sleeplessness. It is often well borne by diabetic patients, even in large doses. A patient sometimes can take 4 to 8 gr. (gm. 0.25 to 0.5), or even more, of opium in twenty-four hours without any bad effects. We have repeatedly administered to patients with a severe form of diabetes, for weeks at a time, from 60 to 100 drops of laudanum, divided into three doses daily, with apparently the best results. It should be especially noted that the bowels were not at all disturbed by the drug. It is deserving of mention that the alkaloids of opium, such as morphin and codein, possess much less value than opium itself. In addition to opium, aspirin in doses of gr. xxijss. to xxx (gm. 1.5 to 2) daily, is worth a trial occasionally, since it apparently diminishes the output of sugar in some cases.

The alkaline mineral waters have gained the greatest reputation in the treatment of diabetes. Hundreds of patients visit Carlsbad, Neuenahr, and Vichy every year, to return much benefited. Of course it must not be forgotten that it is not merely the waters which produce these beneficial changes. Other factors are also important, in particular the regimen, exercise, fresh air, and freedom from the cares of the household and business. Why the alkalies should act favorably we do not know. Perhaps it is because they neutralize the abnormal acids in the organism. Griesinger, and later Külz, as well as others, have made careful comparisons of the amounts of sugar excreted under like circumstances, with and without the ingestion of bicarbonate of soda or of Carlsbad water and similar substances, and for the most part have not been able to perceive any benefit from these remedies. Practical experience, however, shows the value of these alkaline waters; and the use of the above-mentioned resorts is to be recommended, provided the patient is comparatively vigorous and fit for travel, although his expectations should not be raised to too high a pitch. Obese persons with diabetes of a mild degree are best suited for a course of waters at Carlsbad, Neuenahr, etc.

It would serve no purpose to mention now all the other numerous remedies that have been recommended by individual physicians. Not one of them has gained for itself universal recognition. We therefore merely briefly refer to a few "specifics" which have lately appeared, and whose fame is being fostered more particularly by the unscrupulous advertisements of so-called "medical institutions." Such are syzygium jambolanum, dioeat, glycosolvol, antimellin, etc. Upon more careful investigation, the most of these remedies have proved to be without any virtue. Their apparent good effect depends mainly upon the rational diet which is maintained at the same time. A directly favorable action upon the output of sugar can be observed, however, at least in some cases, with syzygium jambolanum.

In impending coma we should above all try the administration of the alkalies. The patient should receive ʒv to ℥j (gm. 20 to 30) of bicarbonate of soda daily, just as soon as a chlorid-of-iron reaction appears in the urine. With the occurrence of symptoms of coma, the dose is to be still further increased. The drug is to be given *per rectum*, or, where possible, intravenously in three- to five-per-cent solution. It is inadvisable to give it subcutaneously, as gangrene of the skin is apt to follow. When coma is fully developed there is usually no hope of rescue. The complications of diabetes, such as tuberculosis, cutaneous affections, etc., necessitate special treatment along the customary lines.

CHAPTER XI

DIABETES INSIPIDUS

Definition and Ætiology.—In the preceding chapter a distinction was drawn between diabetes mellitus and the symptomatic condition termed glycosuria. There is a similar distinction to be made between diabetes insipidus and polyuria. Polyuria is an increase in the volume of urine, and mainly in the amount of water excreted by the kidneys. It is a symptom which may be produced in many different ways. In the first place, it is a natural consequence of the ingestion of large amounts of water, or of the absorption of serous effusions; it also occurs in certain diseases of the nervous system, especially of the medulla and cerebellum; it is occasionally seen, as we have had the opportunity to observe, in chronic hydrocephalus, and is a not very infrequent phenomenon in hysteria. Large amounts of urine are also secreted in certain renal diseases (interstitial nephritis and amyloid degeneration), and often during convalescence from acute diseases, such as typhoid fever, or ofter the ingestion of certain drugs, called diuretics.

Diabetes insipidus, on the other hand, is a disease which may develop idiopathically in persons otherwise perfectly healthy. Its ætiology is unknown. It occasionally seems to be excited by emotional disturbance, concussion or other injury of the brain, or some previous acute disease, such as typhoid or typhus fever, malarial poisoning, and cerebro-spinal meningitis. The disease sometimes appears in the syphilitic, and it may, therefore, in many instances, be due to syphilis. Patients frequently state that their symptoms began immediately after drinking a very large amount of some fluid, as on a very hot day or after a long march. Finally, there are cases in which the disease appears to be hereditary, or to attack members of the same family. A distinction should be made between genuine diabetes insipidus and those cases in which the primary symptom is not polyuria, but an abnormal thirst, polydipsia, so that the polyuria is merely a result of drinking so much water. In many of these cases the polydipsia is undoubtedly a hysterical symptom (see the chapter on Hysteria).

The true nature of diabetes insipidus is entirely unknown to us. The view which seems most probable of any is, that some nervous disturbance is its direct cause. In support of this, we have the appearance of a "symptomatic diabetes insipidus" in connection with organic disease of the brain (*vide supra*), and the fact that polyuria may be artificially excited by injury to a definite spot in the floor of the fourth ventricle or by section of the vagus nerve. Diabetes insipidus presents a most striking analogy to diabetes mellitus. This is shown both by the similarity in ætiology and symptomatology, and still more by the fact that occasionally one disease merges into the other.

Diabetes insipidus is a disease of very infrequent occurrence; and is decidedly less often seen than diabetes mellitus. Most patients are youthful or middle-aged. Genuine diabetes insipidus has also been observed in children, perhaps in connection with hereditary syphilis. The male sex is somewhat more liable to the disease than the female.

Clinical History.—Diabetes insipidus may be developed gradually or with considerable abruptness; the latter case is especially frequent when there is some definite cause, such as the ingestion of a large amount of liquid, or traumatism.

The essential and characteristic symptom is an increase in the volume of urine. This is usually very considerable. Often 8 or 10 quarts (8,000 to 10,000 c.c.) are excreted in twenty-four hours, and cases have even been reported where the amount reached the almost incredible volume of 30 to 40 quarts (liters). If a healthy person and a sufferer from diabetes insipidus are each given the same amount of water in food and drink, the sick man will excrete more urine than the healthy. In color the urine is very pale, and sometimes almost like water. The specific gravity is very low, being usually 1.004, to 1.002, or even 1.001. The reaction is slightly acid, sometimes almost neutral.

The percentage of solid constituents in the urine is, of course, trifling. The total amount of solids, however, corresponds perfectly with the ingesta, or indeed is even somewhat above normal. The amount of urea, in particular, seems to be increased, but it has also been stated that other constituents of the urine have been excreted in abnormally large amounts—namely, phosphoric acid, sulphuric acid, lime, and kreatinin. Inosite has been found in the urine by Strauss and other observers, so that it has even been proposed to give diabetes insipidus the name of "diabetes inositus," in distinction from diabetes mellitus. Inosite is not invariably present, however, in the urine of diabetes insipidus. In cases of true diabetes insipidus, albuminuria is extremely exceptional.

Another important symptom is the excessive thirst. To make up for the loss of water by way of the kidneys the patient is obliged to drink great quantities of liquid, and, indeed, it is always found that the amount of water ingested, in the way of drink and solid food, somewhat exceeds the total volume of urine excreted. Despite this, the tongue is usually dry, as is also the skin, the insensible perspiration being considerably below the normal amount. The furunculosis seen in diabetes mellitus is exceptional in diabetes insipidus. The same is true of pruritus, balanitis, etc. Occasionally profuse salivation has been associated with the disease.

Symptoms referable to the various internal organs are few. Cataract has been occasionally observed, but it is much less frequent than in diabetes mellitus. The same may be said of pulmonary tuberculosis. In most cases the appetite is not excessive. The bowels are regular or slightly constipated. There is seldom much gastrointestinal disturbance, unless from some chance complication. The sexual functions are usually unimpaired.

The general health is considerably affected in cases of any severity. The patient becomes emaciated, languid, and feeble, and has no inclination to mental or physical exertion. Sleep is often disturbed, and the mind depressed. The temperature is normal, or even a trifle below normal, probably as a result of the large amount of cold water drunk.

Diabetes insipidus is a very chronic disease. If there is no serious complication it may last for decades, yet there are cases that run a more rapid and unfavorable course. Sometimes there are considerable vicissitudes in the condition of the patient, in part dependent upon external circumstances and in part apparently spontaneous. In case some intercurrent acute disease develops there may be, during its continuance, a considerable diminution in the amount of urine excreted.

The termination is usually unfavorable. Recovery is extremely rare. In the more fortunate cases the condition finally becomes stationary, and the patient attains to advanced years. Sometimes, however, death occurs more prematurely, being usually hastened by phthisis or some similar complication.

Weil has lately contributed to our knowledge of this disease the results of an accurate study of its hereditary and apparently congenital variety. He narrates the history of a family in which marked polyuria and corresponding polydipsia appeared in numerous members for several generations. These persons all enjoyed excellent health, with this exception; and most of them attained old age. We hardly need to emphasize the fact that this form of the disease is radically different from the ordinary acquired variety. Perhaps its true cause is an abnormal congenital permeability of the glomeruli, but we have no certain information in regard to it.

Post-mortem Appearances.—Such lesions as have been found in diabetes insipidus are usually the result of fortuitous complications, such as tuberculosis, carcinoma, and pneumonia. There are but very few changes referable directly to the disease itself: among these are enlargement of the kidneys and dilatation of the ureters. In rare instances a possible cause for the symptoms has been found in some lesion of the central nervous system, but these were cases of symptomatic polyuria and not of genuine diabetes insipidus. Instances of this sort are seen in connection with tumors or inflammatory changes in the medulla or cerebellum, and exostoses at the base of the skull.

Diagnosis.—The characteristic urinary phenomena usually render the diagnosis of diabetes insipidus an easy matter. It is of course necessary to exclude such diseases as might occasion symptomatic polyuria (*vide supra*); but this is seldom difficult if we make a careful physical examination and duly consider all the attendant symptoms. The differential diagnosis between diabetes insipidus and diabetes mellitus can almost invariably be made by means of the urinometer. If the specific gravity is below normal, it is scarcely necessary to test for sugar, although in doubtful cases this is the sole criterion.[1]

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[1] [Compare, however, page 98 with regard to the presence of sugar in urine of low specific gravity.]

[The early stage of contracted kidney has repeatedly been mistaken for diabetes insipidus.]

Treatment.—No special injunctions with regard to diet are required. It would of course be a mistake to forbid the patient to assuage his excessive thirst; but we may possibly lessen the amount of water drunk by prescribing bits of ice, or lemonade and other acid drinks. Opium sometimes lessens both the thirst and the amount of urine excreted. Rather large doses should be prescribed, say 15 to 20 drops of laudanum, several times a day. It is also important that the skin should be well cared for by means of baths and friction, and every effort should be made to promote the general vigor of the patient. He should have nourishing food and good air.

There have been a great number of internal remedies recommended as specifics, but, in general, we can expect no great results from them. Most employed are valerian, of which 1 to 1.5 drachms (gm. 5 to 10) are given daily in powder, or infusion; and ergotin, of which 1.5 to 8 gr. (gm. 0.1 to 0.5) are given several times a day. Latterly, various observers report good results from subcutaneous injections of strychnin (gr. $\frac{1}{60}$ to $\frac{1}{30}$ [gm. 0.001 to 0.002]), at first on alternate days, later daily. Other remedies which are said to have been sometimes tried with benefit are atropin and the bromids; also, galvanization of the cervical cord.

Occasionally we may find an apparent cause for the disease and endeavor to remove it. If there is a suspicion of syphilis, mercurial inunctions should be tried, perhaps in combination with iodid of potassium or of sodium. Sometimes, as we can confirm by our own experience, they have an excellent effect. Of course, where there is symptomatic polyuria, the primary disease, such as hysteria, demands treatment.

CHAPTER XII

GOUT

(*Podagra*)

Ætiology.—Thomas Sydenham was the first to write a careful clinical description of gout. He himself suffered from the disease for about forty years, and he has given a detailed description of his own case in the treatise which he published in 1683, under the title, "Tractatus de Podagra et Hydrope." It was, however, Wollaston who, in 1797, threw the first light upon the peculiar anomaly of metabolism which exists in this disease. He demonstrated that the gouty deposits found in the joints and other parts of the body are mainly uric acid. From his time an all-important point with regard to the nature of the disease has been the relation between the symptoms of gout and disturbances in the manufacture and excretion of uric acid. In 1848, Garrod showed that in gout the blood contains an excess of uric acid. He was thus in a position to frame a theory consistent with all the clinical facts. Numerous investigations have been undertaken since his day, but we still remain with regard to gout in a position analogous to that which we hold toward diabetes. We are, it is true, in the possession of a considerable number of well-established facts relating to it, but we do not know why the normal chemical processes are disturbed, and we are unable to explain the connection between the various phenomena observed.

Clinical experience has taught us certain remote causes of gout, first of which comes heredity. About fifty per cent of all cases occur in patients whose families have already suffered from the disease, or have given other indications of the uric-acid diathesis—such as calculi, and it is sometimes possible to trace this transmitted taint through many generations. It is decidedly more apt to pass down through the male members of the family than through the female. Besides the direct hereditary transmission of gout, the general family predisposition to anomalies of tissue metabolism in general is, in our judgment, also to be taken into consideration. Occasionally we find obesity and diabetes, as well as gout, in the same family. It should be noted that premature arteriosclerosis also belongs to this related group of diseases.

Next in importance to hereditary influences is the mode of life. From time immemorial this has been regarded as an exciting cause of the disease. It has been a matter of universal belief that overfeeding, and especially the ingestion of too large a quantity of albuminoids, is strongly provocative of the disease. The same opinion has been held with regard to persistent overindulgence in alcoholic beverages. Seneca relates that at the time of the decay of the Roman Empire women practiced such excesses that they were as subject to gout as the men, and an old verse runs: " Wine is the father of gout, feasting is its mother, and Venus is the midwife." That this old view of gout as a rich man's disease has much truth in it cannot be denied. The not infrequent cases of genuine gout which we formerly observed every year in Erlangen attacked mainly great beer drinkers, who for years imbibed five or more liters of beer a day; but, on the other hand, it would be going too far to refer all cases of genuine gout to an improper mode of life, and excessive indulgence in alcohol. Sometimes genuine gouty arthritis is seen in individuals who have always lived moderately, or who have even been in needy circumstances.

Besides alcohol there is another well-known poison which is capable, by its chronic action, of producing that change in metabolism which forms the foundation of gout; we refer to lead. We are absolutely certain that persons who have much to do with lead, such as compositors and house painters, suffer with comparative frequency from genuine gout, with deposits of uric acid in the joints. We have repeatedly seen cases of this sort in Leipsic and Erlangen. In a surprising number of patients lead poisoning is associated with contracted kidney. We have repeatedly observed that the combination of chronic alcoholism and chronic lead poisoning is especially provocative of gout.

With regard to all other alleged ætiological factors, confirmatory evidence is lacking. Possibly, however, when the foundation for the disease is already laid, an attack may be excited by certain determining causes—namely, trauma, taking cold, errors in diet, or mental emotion.

The geographical distribution of gout is remarkably unequal. The frequency of its appearance in England, for example, is universally known. Until recently gout was considered a rare disease in Germany, but this was surely partly due to the frequent failure to recognize the disease. Since I have paid more attention than formerly to the occurrence of gout, the number of cases of gout observed by me has materially increased, and I cannot at all call the occurrence of gout rare either in Bavaria or in Silesia. In Vienna, on the other hand, genuine gout is said to be a very rare disease.

Gout is rarely seen in childhood and youth. It is a disease of middle or advanced life. Men are much more often attacked than women.

Clinical History.—Gout may produce symptoms in many different organs; but its effect upon the joints is so characteristic that the arthritic disturbance has long been termed "normal or regular gout," in contradistinction from "atypical internal gout." This distinction is, of course, an artificial one, for the various phenomena of gout present the most manifold gradations and transitions. It will, however, be advantageous, in attempting to gain a practical insight into the various symptoms of the disease, if we first discuss the so-called "typical attack of gout," subsequently appending a description of the other manifestations of the disease. Furthermore, the regular attack of gout is, in at least a majority of cases (*vide infra*), the first and earliest symptom of the disease.

1. TYPICAL ATTACK OF GOUT.—The typical attack of gout (primary articular gout) is sometimes abrupt, but it is often preceded for a longer or shorter period by certain premonitory symptoms, the meaning of which, though not evident to one who is about to suffer from his first attack, is sufficiently clear to more experienced patients, particularly as each individual case is apt to present a marked similarity in the prodromata of the separate paroxysms. These premonitory symptoms vary in different individuals. Sometimes they consist of dyspeptic disturbances, sometimes of a feeling of languor and mental depression; very often of dragging, muscular pains or cramps in the calves of the legs, or, again, of slight feverishness, with chilliness, sense of heat, and perspiration. These prodromes occur a longer or shorter time before the true attack of gout. On the other hand, it must be confessed that a patient may feel unusually well just before an attack.

The attack is noticeably apt to begin in the night time, or very early in the morning. The patient is awakened by a sudden and very violent pain in the metatarso-phalangeal joint of one of the great toes ("podagra"). The joint becomes swollen, the skin over it red, hot, and tense, the veins in the neighborhood distended. At the same time there is chilliness and moderate fever (101° to 102° F.; 38.5° to 39° C.). This condition persists till morning. Then the pain is almost sure to abate, the fever to remit at the same time that sweating begins, and the patient to feel tolerably well during the day. The joint, however, remains swollen, with inflammation and œdema. The next night the pain begins again, and there is a fresh fever; and these alternations are repeated, as a rule, for three to seven days. Usually there are not many other symptoms present; the appetite is impaired, but ordinarily there are no marked gastric disturbances, such as eructations and vomiting, or intestinal symptoms. There may be a slight bronchitis. Even when the attack is more persistent than this, the pain is usually much less severe after the first two or three nights. After that time it gradually abates, and it is commonly said that an attack is brief in proportion to the violence of its first symptoms. When the pain ceases, swelling soon disappears, the skin undergoes a slight desquamation and resumes its normal appearance, the general health of the patient rapidly improves, and he is often found to be better after an attack than he was before.

For theoretical purposes (*vide infra*) it would be very advantageous to possess a more accurate knowledge of the condition of the urine, and in particular of the excretion of uric acid during the attack and at other times. As yet, however, there have been but few careful investigations made, and these have led to somewhat contradictory conclusions. The assertion first made by Garrod that the amount of uric acid excreted is already diminished several days before an attack, and continues greatly diminished during the attack, has not been confirmed. The excretion of uric acid does, in fact, sometimes seem to be diminished before the attack, but during the attack it is frequently increased. In the intervals between the attacks the amount of uric acid excreted remains approximately normal (His), but even with an unchanged diet the variations seem to be greater than they are in healthy persons. From time to time an apparently spontaneous and not inconsiderable increase in the amount of uric acid excreted occurs. The intentional ingestion of the purin bodies (*vide infra*) generally results in a much more slowly developing and, perhaps also, slighter increase in the uric-acid excretion in gouty subjects than in healthy individuals. The amount of uric acid in the blood is increased during the attack. This is an important fact which was discovered by Garrod, and which has been confirmed by others, including Salomon, Klemperer, and Magnus-Levy. But even at other times than during the acute attacks the amount of uric acid in the blood of gouty persons is generally increased. From $\frac{5}{6}$ to $1\frac{1}{6}$ gr. (gm. 0.05 to 0.07) of uric acid has been found in one liter of blood, whereas in normal blood, at most, only a trace of uric acid is present.

If there has been one attack of gout, there are almost sure to be others. They come sooner or later, at regular or irregular intervals, and separated by weeks, months, or even years. The attacks recur at long intervals in mild cases, more frequently and at gradually diminishing intervals in the severe. Spring and autumn are regarded as the time when attacks of gout are most apt to occur. In these subsequent attacks the great toe still remains the part most constantly and severely affected, but other joints may also suffer—for example, the wrist, the small joints of the fingers (see Fig. 111), the knee, or the shoulder. The distal joints of the extremities are more violently attacked than the proximal. I have never observed gouty disease in the hip joint. In all these joints there appears an acute and very painful inflammation, with considerable swelling of the affected parts. Sometimes traumatism apparently determines the localization of the gouty disturbance. In each attack the trouble is usually confined to a single joint. It is only in exceptional or advanced cases that several joints are simultaneously invaded. Acute gouty inflammations may appear in the tendon sheaths and in the fasciæ, as well as in the joints themselves, especially, for example, in the tendo Achillis, at the outer edge of the foot, etc.

The longer the disease has lasted, the less typical are the separate attacks. There may now be less suffering at the time of an attack, but the articular changes are more persistent. The joints gradually become deformed. There are symptoms referable to other parts of the body, and the gout gradually passes into its second chronic stage. Occasionally the disease is irregular and atypical from its incipiency, and the first manifestations may not be arthritic, but referable to the kidneys (*vide infra*) or other organs.

2. CHRONIC GOUT AND THE GOUTY DISEASES OF VARIOUS ORGANS.—As a rule the most striking results of chronic gout are the changes in the joints. There is a gradual development of marked deformity, particularly in the small joints of the fingers, the joints of the great toe, and less often the other joints. The fingers (see Fig. 112) are deflected toward the ulnar side, at the metacarpo-phalangeal joints, as in arthritis deformans, and at the same time the fingers, as a whole, are thickened, particularly at the joints. Almost always the small finger joints early undergo firm anchylosis. The great toes are often bent out of shape. Not infrequently there are formed around some of the joints which are often attacked, particularly the metatarso-phalangeal joint of the great toe, soft fluctuating masses, which are very painful. If these burst spontaneously, or if they are incised, they are found to contain little, if any, pus, but much detritus, and numerous crystals of uric acid and urates (mainly the monosodium urates). Fig. 113 shows some of these crystals taken from a gouty node of the big toe; on closer examination they turn out to be pure uric-acid crystals. When these nodes break open they may develop into tedious ulcers; similar gouty nodes (arthritic tophi) may develop in other portions of

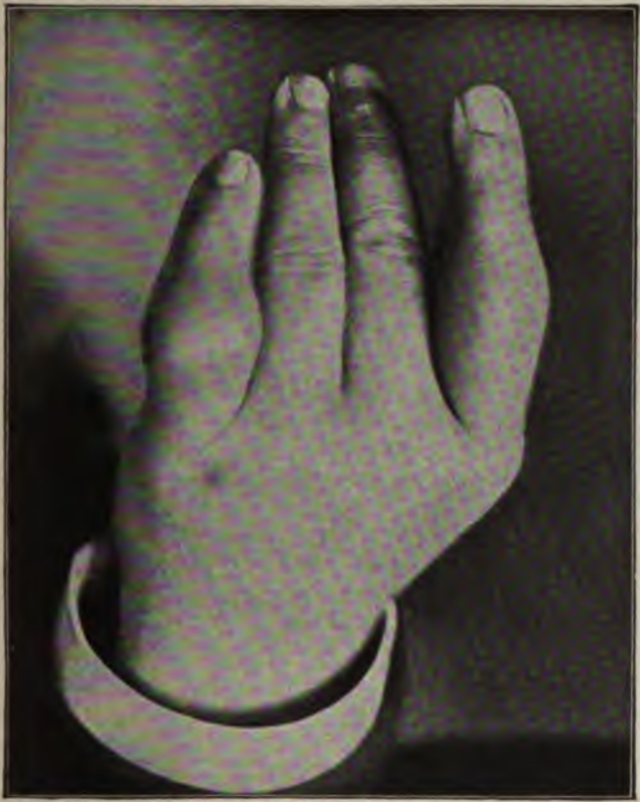

FIG. 111.—Acute attack of gout in the joints of the fingers. (Personal observation.)

the skin, particularly in the lobe of the ear, and sometimes also in the tendons and muscles. The cartilaginous tissues, however, seem to possess a specially strong chemical and physical attraction for uric acid.

Gout may affect the mucous membranes. A gouty dyspepsia is very frequent. Its symptoms are more or less severe. The gouty are also subject to intestinal catarrh of varying severity, and to bronchitis and conjunctivitis, as well as to catarrh of the urinary organs. Ebstein regards "gouty gonorrhea" as essentially a catarrh of the excretory ducts of the prostate gland. It is not an easy matter to explain why these various forms of catarrh should occur in gout. Some of them may be complications. In other cases they are doubtless the result of passive congestion, due to cardiac failure (*vide infra*), but in still other instances it must be confessed that they are apparently due to the toxic influence of the accumulated uric acid or other noxious products of abnormal metabolism.

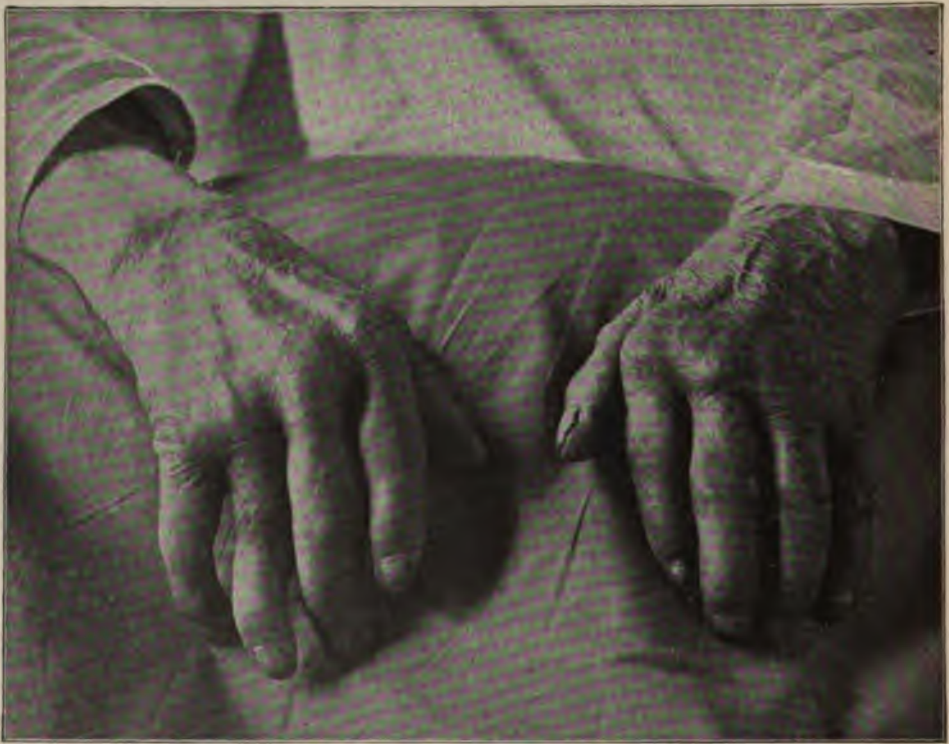

FIG. 112.—Changes in the joints of the fingers in genuine chronic gout. (Personal observation.)

Inflammation of serous membranes—for example, of the pleura—also occurs, and there may be pneumonia. I believe that I have twice observed genuine gouty orchitis. The skin not infrequently suffers from acute or chronic eczema, which sometimes appears to be referable directly to the gout. Keratitis, iritis, and other inflammatory disturbances of the eye are also said to be caused directly by gout. Cirrhosis of the liver has been repeatedly found, and is probably to be referred chiefly, in our opinion, to chronic alcoholism.

By far the most important of all these gouty manifestations are referable to the kidneys and to the circulatory system. The disorder of the latter is sometimes symptomatic of the renal trouble, but in other instances it occurs independently of it. There may be severe gouty arthritis for years without any lesion of the kidneys, but this is exceptional. In severe cases of gout it is the rule that, sooner or later, symptoms of renal disorder present themselves. The so-called "gouty kidney" is a form of chronic interstitial nephritis. However important this complication, its symptoms do not need to be discussed here, as they are precisely similar to those seen in ordinary cases of contracted kidney (*vide* page 770 *et seq.*). The distinctive symptom of this disturbance is albuminuria, and the gradually developing secondary hypertrophy of the left ventricle is the pivot on which turns the whole future course of the disease. So long as the heart remains capable of performing its functions, the condition of the patient will probably remain endurable if not actually comfortable. Finally, however, compensation is sure to become impaired; and then appear œdema, dyspnœa, debility, and emaciation—in short, all the familiar symptoms of cardiac failure. A speedy end may be brought about by uræmia, cerebral embolism, or cerebral hemorrhage; but in other cases the patient suffers for years, both from the heart disease and from fresh arthritic attacks.

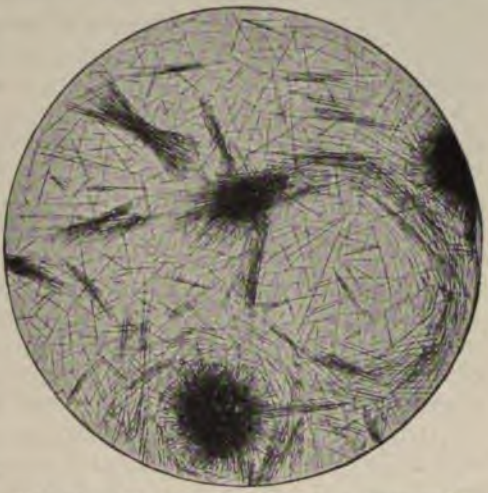

FIG. 113.—Uric-acid crystals from a gouty node. (Erlangen Medical Clinic.)

The cardiac hypertrophy just mentioned is the result of the contracted kidney. Other disturbances of the circulatory system appear to be referable directly to the gout itself. Among these are chronic endocarditis or myocarditis, and perhaps certain "functional" symptoms, such as palpitation and angina. An important symptom is chronic endarteritis or arteriosclerosis. This is often seen in gouty subjects, and in many instances seems to be connected with the gout. Sometimes, also, there are gouty lesions of the veins, such as varicosities or thrombosis. Of course, these changes in the blood vessels may, in their turn, give rise to various disorders.

In a few very rare instances gout seems to attack the brain and spinal cord. Usually, however, the nervous disturbances seen in gout are, as already stated, symptoms of uræmia, or of circulatory disturbance, arteriosclerosis of the cerebral arteries, and the like. The patient may also have certain functional nervous troubles, such as neuralgia or migraine. The direct cause of these is seldom evident.

Complications of Gout.—Apart from chance complications there are several morbid conditions which undoubtedly may stand in close relation to gout. Among these is, first, the formation of renal calculi, or gravel (*q. v.*). This has often been observed in gouty persons. Another not very infrequent combination is gout and diabetes mellitus; ordinarily, attacks of gout appear first, and later glycosuria develops. Often gout and diabetes seem to alternate. The connection between the two affections seems to us to be that both diseases result from the same cause (chronic alcoholism, less often heredity), so that they develop together, or one after the other. Equally obvious is the explanation of the frequent association of gout with obesity, arteriosclerosis, etc.

Anatomical Changes in Gout. Theory of its Nature.—The essential anatomical lesion in gout consists in an abundant deposit of crystalline urates in the tissues. This is most evident in the affected joints, the cartilaginous surfaces of which are often completely covered with white, chalk-like material. In severe cases the same appearance may also be presented by the articular ligaments, tendons, and bursæ, while there are at the same time numerous concretions of urates here and there beneath the skin. These deposits are composed mainly of pure uric acid or of acid sodium urate, with traces of calcium urate, calcium phosphate, and sodium chlorid. However certain it is that these abnormal deposits of uric acid should be regarded as the pathognomonic sign of gout, we are absolutely without knowledge as to their origin and intrinsic importance. One question in particular remains unsolved—whether the deposits of uric acid excite the inflammatory symptoms, or whether, conversely, the deposits of uric acid are secondary and are formed in tissues already attacked by the disease. Ebstein believes that the process of the precipitation of uric acid is invariably preceded by a tissue necrosis. The uric acid while still in solution acts as a chemical irritant upon the cartilage here and there, and thus produces necrosis, whereupon the urates are crystallized out and deposited; but this view has not escaped contradiction, and possibly the primary damage in gout is due not to the uric acid itself, but to some other noxious substance.

The nephritis of gout corresponds in its pathological appearances to that of the true contracted kidney, with one exception; the organs usually present deposits of uric acid or urates, in stripes, both in the lumen of the urinary tubules and also in the epithelial cells and the interstitial tissue. In the connective tissue it is probable that some lesion precedes the deposit.

The lesions presented by the heart, blood vessels, and remaining parts of the body are not especially characteristic of the disease.

With regard to the true nature of gout our knowledge is still more limited and unreliable than in the case of diabetes. Material advance has been made in recent years (Horbaczewski, Kossel, Miescher, and others) only to the extent that we are now, at least, considerably better informed than formerly as to the origin and history of uric acid, which plays the most important, if not the only, rôle in the pathology of gout. We know that uric acid is exclusively a product of nuclein metabolism—i. e., an alteration product of the cell nuclei, and more especially of the nucleinic acid contained in the latter. From this acid the purin bodies (adenin, guanin, hypoxanthin, xanthin) are formed, and from these last, uric acid, which in part is excreted as such, and in part is further decomposed into urea and ammonia. It is probable that these conversions and divisions take place under the influence of definite ferments (Schittenhelm). We distinguish, according to their source, between exogenous uric acid, or that arising from ingested foods (tissues rich in cells, such as liver, sweetbread, etc.), and endogenous uric acid, or that arising from the metamorphosis of the nuclei contained in the cells of the body. Gout seems to be due to disturbances of the entire metamorphosis of the nuclein, which results in an accumulation of uric acid in the blood (uricæmia) and in the deposit of urates in the tissues. It has not yet been definitely determined to what extent uricæmia is due to an increased formation of uric acid, or to what extent it is due to an insufficient excretion of uric acid through the kidneys (so-called "renal gout"). We know just as little regarding the conditions which determine the deposit of uric acid in the tissues. The former assumption of an altered reaction of the blood is not entitled to any consideration. It has been experimentally demonstrated that the urates excite inflammation. The absorption and removal of the urates when tophi disappear is accomplished by phagocytosis.

Diagnosis.—It is usually easy to recognize an acute attack of gout. The sudden onset of the pain and its localization in one of the great toes are very characteristic symptoms, and render it easy to distinguish between it and other acute affections of the joints. When an acute attack of gout affects other joints, we have a sudden development of marked swelling and tenderness in one single place, and a comparatively rapid abatement of the inflammation, while in acute articular rheumatism the disease attacks several joints, and moves quickly from one to another, so that usually the two diseases can be readily distinguished.

As has already been stated, it is almost always the distal joints that are attacked, very rarely the hip joints or the shoulders. The great frequency of the attacks of inflammation of the joints (twenty to thirty and upward) is another characteristic that distinguishes gout from ordinary rheumatic polyarthritis.

The diagnosis is more difficult in advanced stages of the disease, in which the symptoms are more obscure, but in such cases we can usually obtain, by careful questioning, an account of the previous occurrence of typical gouty paroxysms and the existence of certain ætiological factors, such as heredity, mode of life, or chronic poisoning from alcohol or lead, which are of considerable diagnostic importance. In chronic articular gout the main question is its diagnosis from ordinary arthritis deformans. The external appearance of the diseased joints is not of itself decisive, although the gouty arthritis leads to greater and more diffuse enlargement, especially in the fingers, and much oftener occasions complete anchylosis in individual joints than does arthritis deformans. The most important means of diagnosis, however, in every case, are a careful inquiry into the previous history with regard to typical attacks of gout, and a consideration of the characteristic gouty changes —such as tophi breaking open, gouty deposits in the ear, etc. We should also bear in mind, with regard to differential diagnosis, the chronic arthritis of gonorrhea.

If there is chronic nephritis of gouty origin, its source can be recognized only from its association with other indubitable symptoms of gout, particularly typical gouty paroxysms, unless possibly the knowledge of certain ætiological factors, such as a history of gout in the family or of chronic lead poisoning, should put us upon the right track. Ebstein has reported cases of "primary renal gout" where there was no arthritis during the whole course of the disease. Such cases can seldom be correctly diagnosticated during life.

The diagnosis of gout would gain materially in accuracy if it could be based upon demonstrated alterations in the uric-acid tissue metabolism. But much as this is to be desired, it is, at present, generally impossible of accomplishment in practice, for it is difficult to make chemical analyses which could be used, and they can only be of value where due attention is paid to the nuclein intake. The entirely uncontrolled statements as to the amount of uric acid in the urine, which sick persons, unfortunately, nowadays have apothecaries examine for them, naturally have not the slightest value.

From an historical rather than from a practical standpoint, we would mention here the method employed by Garrod to demonstrate the existence of uric acid in the blood. A drachm or two of blood serum, or of the serum obtained from a fly blister, is put into a shallow watch glass, and 6 or 8 drops of a thirty-per-cent solution of acetic acid are added to it. A linen thread is then laid in the fluid, and the whole allowed to remain at a moderate temperature for about a day. If the proportion of uric acid in the fluid is sufficiently large, crystals of that acid will now be found on the thread and may be recognized by their shape and chemical reaction. This "thread test" of Garrod's is not extensively used, because it does not succeed unless there is a comparatively large amount of uric acid in the blood, and, furthermore, uric acid may exist in the blood in health or in other diseases than gout.

Prognosis.—However favorable the prognosis may be with regard to the single gouty attack, yet a permanent release from the disease is rarely to be hoped for. Only such patients as, from the first appearance of gout, pursue most carefully all the requisite prophylactic and dietetic measures, can expect that future attacks will be rare and comparatively mild, and that severe lesions of the internal organs will not occur. So long as the kidneys and other viscera are intact, there is no immediate danger to life, and the patient may attain advanced years despite his gout. The gradual and chronic alterations in the joints may, however, impede locomotion as well as all other movements of the body. Except for this, the general condition of the patient in the intervals between the attacks is often one of tolerable comfort. Indeed, it is frequently the case that the patient will feel at his very best directly after a severe paroxysm, while rudimentary or atypical attacks are often regarded as an ill omen. Really, however, there is no serious danger until chronic nephritis or some other sequelæ, such as diabetes, severe arteriosclerosis, etc., have developed. The prognosis then becomes as unfavorable as in other forms of the diseases enumerated (*q. v.*) and involves the same possibilities.

Treatment.—All authorities agree that the first essential in treating gout is a proper regimen. Unless the patient has energy enough to yield the most implicit obedience to the injunctions regarding diet and mode of life in general, from the first appearance of his disease, no great benefit can be obtained from treatment.

Various authorities have, of late, prescribed special diets for the gouty. The various and almost contradictory theories as to the nature of gout have led to extremely diverse dietetic injunctions. Nevertheless, because of our better knowledge of the origin of uric acid, we are at present in a position to be able to lay down certain rules which are surely not without value. If we cling to simple practical experience, the general conclusion for centuries has been that we should enjoin upon gouty subjects moderation in everything, most of all in alcoholic beverages and in meats. We should advise, therefore, that the total amount of meat eaten should be limited, and that so-called "white" meat (veal, mutton, fowl, fish) should, on the whole, be preferred to "red" meat (beef and game). The cellular organs (liver, kidneys, sweetbread, brain) are now usually forbidden, out of regard to the above-mentioned views as to the formation of uric acid. Milk and eggs may be allowed; fat and carbohydrates are to be given freely, or to be limited, according to the general nutrition of the patient. Gouty subjects who are obese should have less of such articles of diet, while emaciated patients should be encouraged to partake of them. Green vegetables that are easily digested, such as spinach, salad, kohl rabi, cauliflower, Brussels sprouts, and asparagus, have long been regarded as especially suitable. The most appropriate beverages are water, milk, tea, and coffee, also the ordinary mineral waters, Seltzer, Giesshübler, and lemonade. Fruits, especially when cooked, are usually permissible and to be recommended. Such alcoholic beverages as beer and wine are to be allowed in very small quantities, if at all. At all events, everything is to be avoided that might injure the kidneys.

In general, we should not be schematic and theoretical in our dietetic prescriptions, but should consider the individual. The quality of the articles of food eaten is, on the whole, usually less important than the total quantity.

[Many gouty persons, especially those suffering from the irregular form of the disease and acid dyspepsia, are more comfortable and seem to do better on a diet moderate in amount but largely nitrogenous, the starches and sugars being greatly limited in amount. The diet, in fact, should be similar to that laid down for diabetes mellitus, though not so strictly carried out.]

By thus limiting the amount of food taken, we shall avoid any excessive formation of uric acid, and perhaps other harmful substances; but a direct means of promoting metabolism is muscular exercise. If a corpulent patient is still vigorous and is not threatened by any serious internal disease, he should be urged to take a large amount of exercise in mountain climbing, gymnastics, gardening, or similar pursuits. This same indication of accelerating tissue metamorphosis is fulfilled also by bathing. In early stages cool baths and sponging are useful, as are also baths containing common salt, or perhaps even sea bathing cautiously employed. In more advanced cases, particularly if the joints present permanent lesions, the warmer baths are more desirable, such as are found in Wiesbaden, Baden-Baden, Teplitz, Ems, and Aix.

The internal administration of alkalies is an efficient adjuvant to the dietetic and hygienic prescriptions above enumerated. Although there may be doubt about the correctness of the ordinary theoretical basis for this prescription—viz., that alkalies prevent the deposit of uric acid in the tissues—yet simple practical experience justifies the employment of suitable mineral waters. These waters stimulate the conversion of the albuminoids, increase the activity of the kidneys, benefit gastric catarrh, and overcome constipation, and in all these ways combine to produce a favorable effect upon the patient's health. Still another factor is the judicious diet and mode of life at such health resorts. The waters of Carlsbad and Vichy have gained the greatest repute in gout, although the waters of Ems, Neuenahr, Fachinger Wasser, and Kronenquelle have an analogous composition, and doubtless they would produce similar effects. Most of these waters can be taken very well in the patient's own home. The salts of lithium especially promote the solution of uric acid, and the waters which contain lithium have been strongly recommended. The natural springs of this sort, such as the Crown Spring in Obersalzbrunn, and those of Assmannshausen and Salzchlirf, contain comparatively insignificant amounts of lithium; and it might be a better way to use the artificial lithium waters, such as are made by Struve or Ewich. Another way is to prescribe carbonate of lithium, in powders containing 2 or 3 gr. each (gm. 0.10 to 0.20), one powder two or three times a day, in a glass of Seltzer or Biliner water.

As to the treatment of the acute attack, it has ceased to be customary to employ any potent remedies. The patient must, of course, keep his bed. The affected joint should be wrapped up in cotton wool, the whole leg elevated, and a proper diet strictly enjoined. Free movement of the bowels should be maintained by means of enemata. If there is considerable gastric disturbance, bicarbonate of sodium, magnesia, or some bitter may be prescribed. The most certain remedy for severe pain is a subcutaneous injection of morphin. Less efficient are narcotics, locally applied, and warm compresses. Whether any internal remedies are calculated to abbreviate the attack is doubtful. Formerly colchicum (20 or 30 drops of vinum colchici seminis three or four times a day) was the favorite medicine, but it seems to be going out of use.

Colchicin may be used cautiously (pills of gr. $\frac{1}{30}$ = gm. 0.002 each [colchicin, Merck] two or three times daily). Salicylic acid and salicylate of sodium may be administered in the same way as in acute articular rheumatism, and sometimes, although not always, they are followed by improvement. We have several times seen quite good results from aspirin in doses of 10 to 15 gr. (gm. 0.5 to 1) several times daily. Antipyrin, salipyrin, salol, and similar remedies sometimes succeed in allaying the pain in the joints. Some physicians recommend subcutaneous injections of antipyrin in the neighborhood of the joint, but we have no personal experience on this point. On the basis of dubious theoretical assumptions, a number of drugs have been recommended recently which possess the power of dissolving large amounts of uric acid. Prominent among these are piperazin, of which about 8 gr. (gm. 0.5) are given daily in soda water; lysidin, 15 to 75 minims (1 to 5 c.c.) of a fifty-per-cent solution daily; sidonal (a combination of cinchonic acid with piperazin), gr. xv (gm. 1), in powders five or six times daily, etc. Many physicians report favorable results with these remedies; others, on the contrary, have found no virtue in them.

[One reason why colchicum has fallen into relative disuse is that the relief obtained from it is often so prompt and complete that patients are tempted into continued indulgence in a faulty manner of life. It is also supposed by some that the drug interferes with the effort of nature to eliminate the poison, which becomes generally diffused, and sets up changes in the vessels and internal organs. Precisely how colchicum acts we do not know, but that it does act, and sometimes with marvelous success, there can be no question.]

The chronic affections of the joints in gout are treated as are other varieties of chronic arthritis. The most efficient remedies are cautious massage and baths, including hot sulphur baths and mud-and-sulphur baths, also Bier's hyperæmia and hot-air treatment. The internal administration of the alkalies, lithium, and similar drugs, to combat the general gouty diathesis, should be associated with these external remedies. Some physicians report that iodid of potassium favors the absorption of the gouty deposits.

The treatment of the nephritis and other complications need not be discussed at length. The gout itself should always be the main object of our therapeutic efforts, and beyond this we may be guided by general principles.

CHAPTER XIII

OBESITY

(Corpulence. Polysarcia adiposa)

Definition and Ætiology.—The amount of adipose tissue in the body is subject to considerable variation, and it is not possible to state absolutely what should be considered as normal and what as abnormal. For practical purposes we may draw the line where the increased size grows burdensome to the individual. After a certain point, any further addition to the amount of fatty tissue is almost sure to work serious injury, and is therefore to be regarded as an actual disease, and not merely an inconvenience. It should be said, however, that in such cases the symptoms of obesity are very frequently confounded with those springing from other disorders—these latter possessing, indeed, the same ætiology as obesity, but distinct from it.

The most frequent and important cause of obesity is the habitual ingestion of too large an amount of food.

A prime factor in the increase of adipose tissue in the case of the well-to-do, is insufficient muscular activity, coupled with an abundant ingestion of nourishment. That an increase of adipose tissue may arise from overnourishment even in spite of a considerable amount of muscular activity, is shown by the obese Bavarian brewers and butchers, who, despite their hard labor, cannot entirely dispose of the quantity of carbohydrates introduced into the system through their excessive beer drinking. It is a matter of indifference whether the excess is composed mainly of albuminoids, carbohydrates, or fats; for in either case, if the quantity be sufficient, an increase of adipose tissue may take place. We shall soon see, however, that the excess is usually in fat and carbohydrates. Inasmuch as this overfeeding is almost certain to be habitual, the excess at any one time need not be at all large. We often hear a corpulent person express his surprise that he grows heavier every day, although he "does not eat any more than others who are lean." The explanation is easy if we consider that a daily increase of 5 gm. of fat (1.025 drachm) suffices to increase the weight in ten years—for example, between the thirty-fifth and forty-fifth years—40 pounds avoirdupois. In reality the daily increase is sometimes greater than this.

The basis for a detailed consideration of the causes which lead to the deposit of fat will be gained by a consideration of the physiological laws relating to nutrition discovered by Voit, Pettenkofer, and their pupils. It has been shown that both the albuminoids and the carbohydrates of the food may be a source of fat, formed within the economy, and also that the fat contained in the food may be, to a large extent, directly deposited in the fat cells of the body. One product of the decomposition of albuminoid substances is invariably fat. This usually undergoes further oxidation, but it is sometimes retained unaltered in the system. It would even seem that the albuminoids give rise to much more of the fatty tissues of the body than are produced from the carbohydrates ingested, although there is no doubt that these latter may also yield fat. Carbohydrates do promote obesity, but less because they are a direct source of fat than because they are easily decomposed, and so shield both the ingested fat and that which is formed out of the albuminoids from oxidation. In this indirect way they do greatly favor a tendency to corpulence.

We thus perceive that various diets may, each one of them, permit of an increase of adipose tissue. In actual life, of course, the most frequent conditions are such as result from the customs and habits of the population in general. The diet is almost invariably a "mixed" one—that is, it contains albumen, fat, and carbohydrates—and in most instances the obesity is due to an excessive amount of all three elements, or at least of the fat and carbohydrates. A person may, however, become corpulent who eats very little fat, provided he consumes a large quantity of albuminoids and carbohydrates; or, again, if he eats very little starchy food, provided he consumes a large amount of meat and fat.

Perhaps these facts may be made clearer by giving a concrete example. Voit tells us that a vigorous man who requires daily 118 gm. of albumen and 259 gm. of fat to maintain a physiological equilibrium as regards fat and albumen will, other things being equal, begin to store up fat if there is any further increase in the amount of fat in his diet. The same result will also take place if, instead of the rations previously stated, he ingests more than 118 gm. of albumen and 600 gm. of starch, or more than 664 gm. of albumen alone, or, finally, more than 118 gm. of albumen, 100 gm. of fat, and 368 gm. of starch.[1] It is obvious that this last diet, which closely resembles the average diet of an adult[2] in good circumstances, whose weight is neither increasing nor diminishing, is the one most likely to be exceeded; whereupon there must take place a deposition within the system of the superfluous fat.

Among the various kinds of food is one group which deserves mention as being an important factor in many cases of obesity; we refer to alcoholic beverages. There can be no doubt that intemperance in this regard plays a prominent part in many instances. Sufficient illustration is furnished by brewers, hotel keepers, and the inhabitants of countries like Bavaria, where beer drinking is prevalent. In this particular it is evident that beer works more harm than does wine or strong liquor; for beer contains, in addition to the alcohol, an appreciable amount of starchy material, making the sum total from the beer drunk during the entire day a considerable one. Many persons who would be extremely indignant if called "tipplers" habitually take five or six glasses of beer a day, equivalent to 150 gm. of starch, or, in other words, to one half the total amount of starch required by the system. Even this quantity is frequently exceeded. Of course the three or four per cent of alcohol which the beer contains also promotes the deposition of fat. Alcohol is readily oxidizable, and it thus shields to a considerable extent the fat already present in the body (1 gm. of alcohol furnishes on oxidation 7 calories); and it also, in all probability, works such injury to the tissues as to diminish their power of effecting decomposition. We must also remember that most beer drinkers take little exercise.

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[1] This statement is founded upon an important discovery of Rubner, that, as far as the storing up of fat is concerned, the measure of value for different foods is the amount of heat given off by them when they undergo oxidation into carbonic-dioxid gas and water. Measured in this way, 100 gm. fat = 211 gm. albumen = 232 gm. starch = 234 gm. cane sugar = 256 gm. grape sugar.

[2] Probably the amount of fat contained in the food is often less than the above, and the amounts of albumen and starch somewhat larger. Voit estimates the diet of a well-to-do person at 127 gm. albumen, 89 gm. fat, and 362 gm. starch; and that for a vigorous laborer at 118 gm. albumen, 56 gm. fat, and 500 gm. starch. Of course, these figures are merely approximate. Among the poorer classes the amount of albumen, especially in the food, is materially less. Reckoning by calories, the average metabolism of an adult male amounts to 2,400 to 2,800 calories; with continual muscular labor, this conversion at once rises to 3,000 to 3,500 calories. Hard-working porters convert as much as 5,000 calories and over.

The long sitting at the beer table, and the mental and physical sluggishness which immoderate indulgence in beer always causes, and, finally, the increasing corpulence itself, explain why most beer drinkers dislike protracted exercise.

We have thus seen that in by far the larger number of cases obesity is mainly due to the ingestion of too much food. No weight need be attached to the usual statement of corpulent persons, that they eat no more than others. Few of them have any idea how much nourishment they do consume. Others, having already become corpulent, eat less, to be sure, than they used to, but nevertheless an amount sufficient to maintain the acquired weight.

Other factors may, no doubt, exert an influence upon the increase of adipose tissue by diminishing the consumption of fat in the system. The most important factor of this class is physical inactivity. Muscular contractions lead to the decomposition of a large amount of fat. This explains why people of sedentary habits, and those who sleep long and exercise little, are more apt to become corpulent than are manual laborers. Undoubtedly, most of the cases of abnormal obesity arise from the well-known frequent combination of superabundant nourishment with limited muscular activity. The scantiness of the muscular labor performed does not always depend upon social conditions or physical indolence. Other morbid conditions, which, while not interfering with food consumption, prevent any extended muscular activity, often lead to abnormal obesity. In patients suffering from chronic joint affections, paralysis, heart disease, certain cerebral defects and the like, we frequently find pronounced corpulence, which undoubtedly is referable to bodily inactivity.

The question as to the origin of obesity becomes much more difficult if we attempt to determine whether, besides this "simple obesity from overfeeding," there is also a constitutional obesity—i. e., an obesity that cannot be referred to an excessive ingestion of food, but which arises from a pathological impairment of the body's power to dispose of the fat introduced into it. This question can only be decided by means of very difficult and laborious examinations into tissue metabolism in obese subjects. So far as can yet be inferred from such examinations, there really seems to be a diminished power of fat combustion in some obese persons. The cases of corpulence in young children, its appearance as a family or an hereditary characteristic, the tendency of certain races (the Hebrew, for example) to obesity, may all be cited for the establishment of such "constitutional" conditions. In all these instances, the importance of a "tendency" to obesity should not be overrated. Upon careful investigation we shall almost invariably find in the habits of the individual, as regards food and exercise, a satisfactory explanation of his obesity. At all events, we are acquainted with a series of physiological facts from which it would seem that the amount of fat converted is dependent on certain internal conditions.

Thus we know specially that the combustion of fat is unquestionably increased by the ingestion of thyroid-gland substance, and that, accordingly, obesity has frequently been found associated with abnormal smallness of the thyroid gland. We know, in addition, that castration leads to abnormal corpulence in males as well as in females. This has been confirmed by experiments, and undoubtedly stands in relation to a number of clinical observations (increase of fat after the menopause, after gynecological operations, the infantile form of adiposity, etc.). At all events, we have here a very interesting subject that needs further investigation.

Pathology.—After corpulence has passed a certain point the condition is evident at the first glance. The subcutaneous cellular tissue is one of the chief places in which the fat is deposited. Consequently, the panniculus adiposus soon attains considerable thickness. The countenance grows more round and plump; beneath the chin is formed a second prominence known as the "double chin," the chest appears broadened, the waist enlarges, and, particularly in women, the breasts are changed to great shapeless masses, over which the skin is so tightly stretched as to present lineæ albicantes. The abdominal walls are greatly altered. The belly projects more and more until it becomes actually pendulous, and its lower surface touches the interior surface of the thighs. Intertrigo is apt to occur in the groins, below the breasts, and between the buttocks. The skin everywhere has a fatty feel, due to the increased secretion of the sebaceous glands. This hyperplasia of the fatty tissue in the panniculus adiposus is associated with a deposit of fat in many parts of the interior of the body, including the mesentery, mediastinum, pericardium, and the capsules of the kidneys. Some of these will be mentioned again farther on.

Of course the circumference and weight of the body become greatly increased. As an approximate measure it may be stated that for men of middle height a weight exceeding 90 kgm. (200 pounds), and for women 75 kgm. (165 pounds), may be regarded as abnormal.[1] This increase in bulk is the first cause of subjective symptoms. An obese person has to exert a greater effort in making any motion than do other people, and as a necessary consequence he gets easily tired, and seeks as far as possible to avoid exertion. The increased demand upon the muscles explains the familiar fact that corpulent persons perspire readily.

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[1] A weight of even 150 kgm. (330 pounds) has been repeatedly observed.

The graver symptoms of obesity, and properly the first pathological phenomena of the condition, relate to the respiration and the circulation. The patient begins to complain of shortness of breath and is subject to marked dyspnœa upon running or climbing stairs. There may be, at the same time, cardiac disturbance, indicated by a rapid pulse, palpitation, intermission of the pulse, or other slight irregularities in cardiac action. All these symptoms grow gradually worse, and to them are added symptoms of cardiac failure and consequent passive congestion. There is a tendency to bronchitis and other catarrhal troubles. The appetite and digestion are affected, and œdema appears.

A careful analysis of all these symptoms shows that many causes combine to produce them, all having a common tendency to impede respiration and, still more, circulation. One source of disturbance is the increased deposit of fat upon the framework of the body. It is probable that the excess of adipose tissue covering the thorax exerts a direct influence in obstructing the respiratory motion of the thoracic walls, and renders the respiration more superficial. In this way the return of venous blood to the heart and the pulmonary circulation are both impeded, because the negative pressure in the chest is less than normal. Likewise the diminished amount of bodily exercise affects the circulation unfavorably. Brauner has shown how numerous are the arrangements connected with the fasciæ for promoting the venous currents, by means of the negative pressure resulting from the movements of the body. Whether the fat deposited around the heart directly obstructs the cardiac movements is somewhat doubtful, though this view is held by many. The fatty infiltration of the myocardium is of more importance: the fat is deposited in the intermuscular connective tissue. Still, it is doubtful whether this condition, which is not so very frequent, after all, occurs as a primary change, or whether it is not rather a result of previous atrophy of the mycardium (compare the chapter on Fatty Heart).

There is no doubt that, in almost all cases where the corpulence actually induces grave disturbance, the cardiac symptoms are of prime importance. These are due, as has just been indicated, in part to the increased amount of adipose tissue, and in still greater part to complications, most of which are excited by the same causes as is the obesity itself. The abundant adipose tissue may obstruct the circulation in the smaller blood vessels and capillaries inclosed within it. Furthermore, the excessive development of fatty tissue probably leads to the growth of new blood vessels, and, as a consequence, to an increase in the total volume of the blood. This is one way in which the demands made upon the heart are rendered greater than normal, and explains why the corpulent frequently exhibit cardiac hypertrophy. Other influences are also at work to produce this same result: they are, in the first place, the same factor which occasions the obesity itself—namely, the ingestion of increased amounts of food and drink (see the chapter on Cardiac Hypertrophy), and, secondly, certain other lesions which are frequently associated with obesity and are referable to the same causes. Chief among this latter class is general arteriosclerosis. If this involve the coronary arteries, it may in turn occasion still further damage—for example, degenerative myocarditis.

Chronic interstitial nephritis is another not infrequent complication. This is referable in part to the same causes as is the obesity. Gout and diabetes are less frequent.

We thus see that obesity is often merely one of many injurious results occasioned by an improper mode of life. It is, in a certain sense, the first danger signal warning the patient and his physician to avoid the graver disturbances which threaten. This is a point of great practical importance; for, when once we have a combination of obesity with cardiac hypertrophy, fatty infiltration of the heart, arteriosclerosis, or interstitial nephritis, the various causes and effects act and react upon one another in a way most perilous to health, and even to life. There is no need of describing the grave disturbances which invariably develop at the close of the scene. They are the result of chronic cardiac insufficiency, and have been fully described under cardiac disease.

In every case of obesity the physician should examine the heart, lungs, vascular system, and kidneys, particularly if there be already subjective disturbances. The examination of the heart may present considerable difficulties, because the results of palpation and percussion are so much obscured by the thick cushion of fat which covers the thorax. We can, however, have recourse to auscultation, and can feel the pulse. The pulse may be either rapid, slow, or irregular. In the more serious cases an X-ray examination should never be omitted. We need not mention any minute particulars as to the examination. It may be stated, however, that a hepatic enlargement is often found, but it is much less often the result of fatty infiltration than of simple hypertrophy or passive congestion.

We have thus seen that corpulence may sometimes be associated with grave and dangerous lesions; but, on the other hand, it should be stated that this unfortunate condition by no means invariably exists. Not infrequently the corpulence remains moderate, in which case it is not really dangerous, however inconvenient. This is true of a proportion of those cases which are due to the ingestion of a large amount of nourishment, associated with defective oxidation, and where there are no other unfavorable influences at work. The obesity of hard drinkers is almost always a more or less dangerous condition, while that seen in many elderly persons and in women is often comparatively free from peril. These latter individuals are, to be sure, discommoded by their great weight; they can accomplish less than they used to—they are easily put out of breath, and have a certain tendency to catarrhal inflammations and rheumatic difficulties, but they escape the severer lesions above enumerated. Even these apparently harmless conditions should not be disregarded by the physician, as he can never be absolutely certain that grave complications may not be developed eventually.

In time we shall probably succeed in more exactly defining the various forms of adiposis and their signification. Certain clinical types already begin to receive separate recognition. Thus, for instance, many cases of extraordinary adiposity in children of from ten to fifteen years of age bear a striking resemblance to one another. We refer especially to those cases of boys, mostly round-headed, not particularly well-grown, but rather intelligent, who present an unusually strong adipose development on the trunk and the haunches. The abnormal smallness of the genital organs (small penis, very small testicles), which have remained entirely child-like, is striking. Analogous cases, also with anomalies of sexual development, occur in girls. In this connection we may briefly mention adiposis dolorosa, described by Dercum. This consists in a remarkable symmetrical lipomatosis of the extremities and the body, but not involving the hands and feet, and is associated with severe pains, as well as marked hyperæsthesia of the skin and the deeper soft parts. This affection is frequently observed in elderly women with a constitutionally nervous predisposition. We would here also briefly refer to the peculiar cases of circumscribed multiple symmetrical lipoma.

Treatment of Obesity.—To cause the disappearance of the accumulated fat, it is necessary to promote its oxidation in the system and to prevent the ingestion of new supplies of fat. To accomplish this purpose we possess only two means—first, a limitation of the ingestion of such kinds of food as may lead to the formation of fat in the system, and, secondly, stimulation of muscular activity and other factors which occasion the destruction of the fat already present. All the various methods of treating obesity, without exception, aim either to diminish the supply or to increase the destruction of fat.

The methods vary greatly. It must be borne in mind that the diminution of the adipose tissue must not involve injury to the body itself. The treatment should not weaken the constitution, but should invigorate the patient, or at least be innocuous.

It is of prime importance, in every method of treatment, that the total quantity of ingested food should be diminished. It is a mistake to forbid the patient some particular kind of food—for instance, the carbohydrates or the fats—with the idea that they alone do harm, or to allow him unlimited quantities of other kinds of food, in the belief that they are harmless. Any person can eat albuminoids, fat, and starch at every meal, and yet not grow fat; while, on the other hand, too much of any one of these may lead to an increase of adipose tissue. The amount of food which a person can take without increasing the amount of adipose tissue varies with the individual. It depends upon the amount of material already present in the body, and upon the various demands made upon the system, as well as other factors. This renders it difficult to draw up a universal dietary for the obese. We can best judge of the value of any course of treatment by its results, and these are best measured by the weight and the subjective condition of the individual patient.

Of the various elements of food, the albuminoids should be diminished least of any, because it would be sure to work injury to the system if they were supplied in too small an amount. Of course, the albuminoids must not be eaten to such an extent that the fat into which they decompose remains intact in the body. An increase in the amount of nitrogenous tissue is, however, desirable, because this promotes the vigor of the muscles and the heart, and so leads to the oxidation of larger quantities of the nonnitrogenous tissues.

The amount of fat and starch must be much more limited. The fats and starches are more potent in increasing adipose tissue and in shielding from oxidation the fat already stored up in the body than is nitrogenous food. It would not be at all advisable to forbid the use of either one of these two constituents of a mixed diet, allowing the other alone to be eaten. A varied diet is extremely desirable, even for one who is corpulent; and we should exclude neither fat nor starch wholly from the dietary, but we should merely limit the amounts to be taken. As already indicated, the amount of albuminoid food remaining unchanged, a person can eat double the quantity of starchy food that he can of fat, without increasing his adipose tissue. It is therefore irrational to allow the corpulent fat in larger proportions than starchy foods. The diet which Ebstein has recently proposed for the treatment of obesity does prove successful, but the explanation of its success lies in the comparatively small amounts of meat and fat ingested. Precisely the same results would be attained if a corresponding amount of starch were substituted for all or a portion of the fat; and in practice it is desirable, at least in most cases, to allow the patient both starches and fats. Of course, the likings and experience of the individual should be considered in each separate case. The Banting treatment, introduced in 1864, enjoyed for a time a great reputation. Its inventor applied it first of all to his own case, and with success. It rests upon a rational basis, inasmuch as the albuminoids are allowed in abundance, and the ingestion of fat and starch is limited. It lays too much stress, however, upon the exclusion of fat as compared with starch. Of great practical importance in many cases is the strict prohibition of beer drinking. Many cases of obesity are due merely to the excessive amount of carbohydrates contained in the unreasonable quantity of beer drunk daily. If corpulent beer drinkers of this sort change their mode of life in this single particular, and drink no beer whatever for from three to six months, they are almost sure to lose a great deal of weight.

The physician who bears in mind the principles just expounded can lay down his own rules for the diet of his patient. As already stated, it is impossible to give figures which will apply to every case.

There is no one method of removing obesity that is to be particularly recommended. The same end can be attained by the most diverse methods, and the skill of the physician consists only in so arranging the diet as to attain the desired effect without injury, and without causing too much discomfort to the patient. If we take as a basis the average diet for an adult—that is, about 125 gm. of albumen, 80 gm. of fat, and 350 gm. of starch—we might say that most cases of obesity would be sure to undergo improvement upon a diet containing 125 gm. albumen (or possibly even more than this), 40 gm. fat, and 150 gm. starch. The amounts of fat and starch could be even more diminished, but it is usually best not to be too precipitate. A gradual diminution of 2 or 3 pounds a week, extending over a long period without interruption, is to be preferred to the rapid treatment common at many health resorts. Of course, the loss of fat is greater at the commencement of treatment than later on, when the amount of adipose tissue has already approached more nearly to normal, and the diet must undergo a gradual and corresponding change. It is of particular importance to increase the amount of nonnitrogenous foods in the later stages of treatment, lest the albuminoid tissues of the body become wasted.

The following dietary may be taken as an illustration of what would be suitable for a patient in the beginning of treatment: For breakfast, a cup of coffee with milk, and about 75 gm. of bread. At noon, a plate of soup, 150 to 175 gm. of lean meat or fish, lettuce, green vegetables, and about 25 gm. of bread. For dessert, about 75 gm. of boiled rice, or some simple pudding, or 100 gm. of fruit. To quench the thirst, water, or half a pint of light wine. In the afternoon, a cup of coffee, and with it not more than 20 to 30 gm. of bread. For supper, 2 eggs, or 100 to 120 gm. of meat, with 30 gm. of bread, a little fruit, lettuce, ½ pint of wine, or 1 or 2 cups of tea, not much sweetened. Butter should be entirely proscribed at first; later on it may be used in small amounts.

Some approach to this bill of fare must be enforced not merely for a few weeks, but for months. It is absolutely necessary that the patient should be weighed every two or three weeks. If the weight diminishes slowly and gradually, without any subjective disturbance, we have the best proof that the diet is a suitable one. If the weight does not diminish, then the amount of ingesta must undergo further reduction. If more food can be taken without the weight increasing again, a larger amount may be unhesitatingly permitted, and indeed may even be advisable if the patient be languid. The increase should at first, however, be mainly in the amount of albuminous food, the amount of starches and fats not being much increased. The "cure" cannot be regarded as complete until the weight has been brought down to that of the average individual of the given age and sex. This goal having been reached, greater freedom in diet is permissible.

The object of the treatment just suggested is exclusively the limitation of the production of fat. We may also promote the destruction of the fat already stored up in the system. A chief means to this end is muscular exercise, which undoubtedly increases the oxidation of the adipose tissues. Carried out in a proper manner, it is therefore a most valuable adjuvant to treatment. Oertel has pointed out that muscular exertion does good in still another way—namely, by promoting cardiac activity and inducing deep respiratory efforts. Thus the heart is strengthened and circulation promoted. Mountain climbing is one of the best modes in which to take the desired exercise. We need hardly say that the increased muscular activity makes it possible for the patient to take an increased amount of food without injury. In dealing with cases of obesity in which the condition is due less to overfeeding than to lack of muscular exercise, it might be decidedly advisable to lay the most stress upon the increase in muscular activity; for certainly it is more advantageous for the organism to get rid of its excess of fat by means of lively metabolism, while nutrition is well maintained, than to be more or less starved by an excessive limitation of diet. We should not rest satisfied with a single mountain tour, but should insist upon a persistent and considerable employment of the muscles in gymnastics, walking, and the like. The different forms of apparatus for home gymnastics that can be purchased, such as those for gymnastics, rowing, etc., are very useful.

Baths also promote oxidation, but they are far less potent than is muscular exercise. Cold baths, brine baths, or baths containing carbonic-acid gas, may be employed. One way in which they do good is by stimulating the nervous system. Oertel regarded it of great importance to diminish the amount of water in the system. Although this point of view is of importance chiefly in the treatment of circulatory disturbances (see Vol. I), because it is claimed that the mechanical conditions in circulatory disease and passive congestion are much improved by a diminution of the amount of water in the body, still, this factor is not to be wholly disregarded in the treatment of obesity; but the chief cause of the rapid loss of weight, which we actually see in the corpulent as a result of the deprivation of fluid, is probably to be found not in the loss of fat, but in the marked loss of water contained in the body. We must also remember that in many cases of obesity the limitation of drinking includes the restriction of beer drinking. "Desiccation" may further be promoted by stimulating the perspiration by bodily exercise or by steam baths. This withdrawal of liquid from the system, however, is advisable only in cases where there is already incipient cardiac failure.

In conclusion, we have still to mention the treatment of obesity by thyroid-gland preparations. That the administration of these increases tissue metabolism, and that the weight of the body may thereby be decreased, is beyond question. But in spite of this, the treatment is not in general to be recommended, as these preparations induce not only a loss of fat, but also a decrease of body albumen, and the ensuing loss of weight is therefore often associated with a not inconsiderable deterioration of the general condition.

Only in those isolated cases in which we are justified in assuming a constitutional obesity that has some connection, perhaps, with a defective activity of the thyroid gland (*vide supra*) is a trial of thyroid-gland treatment justified, and sometimes successful.

It is evident that numerous excellent methods are at our disposal for the treatment of obesity, but their application to any particular case should be the result of a careful consideration of the special circumstances presented. A very essential point is that the injunctions of the physician should not merely be given, but be carried out, and it is precisely here that the treatment of many cases suffers shipwreck. We may be baffled by the patient's lack of energy and persistence, or by the importunate demands which his profession or social position make upon him. Indeed, it is sometimes absolutely impossible to prosecute the treatment at home, in which case bathing and health resorts are to be urgently recommended. There alone can the patient muster up the resolution necessary for carrying out the desired changes in his mode of life. The incontestable success of treatment at Carlsbad, Marienbad, Kissingen, Tarasp, and similar resorts is doubtless only to a very small extent the result of their specific medicinal influence, but it is mainly due to a strict observance of the above-described diet and regimen. The internal use of mineral waters is not entirely without a beneficial effect. Their laxative qualities diminish the absorption of food from the intestinal canal. It should be said, however, that the patient is at the same time exposed to the danger of a waste of his nitrogenous tissues. This is why patients frequently complain of the debilitating effect of these mineral springs; to avoid which, it would be well to increase the amount of albuminoids in the diet. We must also consider the need of guarding against too great a supply of fluid, with regard to what was said above as to the occasional advantage of "desiccation" of the body.

CHAPTER XIV

SCROFULA

Definition and Symptoms of What is Called Scrofula.—We desire to present, at the close of this section, a brief description of scrofula, but merely from a practical standpoint. From a scientific point of view scrofula is not to be regarded as any special variety of disease. The term is applied to a group of symptoms seen most frequently in childhood, the essential features of which consist in the appearance of chronic enlargements of the lymph-glands, and in certain diseases of the skin and mucous membranes. The simultaneous appearance of these various phenomena does really produce a somewhat characteristic picture, which can frequently be recognized at the first glance.

Most scrofulous children appear pale, with a flabby skin and soft muscles. The panniculus adiposus may nevertheless be tolerably well developed. Not infrequently the face is puffy, with prominent lips. This is called the "torpid habitus." In other cases the child has small features and a remarkably delicate white skin, which but partly conceals the superficial veins, and is readily suffused with blushes. To these the name "erethitic habitus" is applied.

Enlarged lymph-glands are to be felt in the throat, at the angles of the lower jaw, and in the back of the neck, and occasionally in other parts of the body. These glands may remain indolent for a long while, or they may suppurate and break externally. Chronic cutaneous eruptions are often seen in various places. The most common of these is a scaly or impetiginous eczema, affecting the face, scalp, or extremities. More severe affections are lupus (*"lupus scrophulosorum"*), prurigo, and lichen scrophulosorum.

Of the mucous membranes, the conjunctiva and the lining membrane of the nostrils are most frequently affected. Conjunctivitis in various forms is a characteristic symptom of scrofula, as are also blepharitis ciliaris, keratitis, and chronic rhinitis, which last often terminates in a pronounced ozæna (*q.v.*). Chronic diseases of the ear are also frequent, such as otitis media, with perforation of the tympanum, and occasionally caries of the mastoid cells and its unfortunate results.

Of the deeper-lying tissues, the bones and joints are most apt to suffer. The affections located here are almost exclusively "fungous"—namely, fungous ostitis and periostitis, white swelling, and caries. Formerly there was frequent use of such terms as "scrofulous inflammation of the knee joint," or "scrofulous caries of the ribs."

If we inquire into the nature of this strange group of symptoms thus briefly enumerated, we shall find that by far the greater number of cases of well-marked scrofula are examples of tuberculosis. Tubercle bacilli have been demonstrated in connection with most of the fungous or "scrofulous" diseases of the bones and joints. Ozæna is often a tubercular disease of the nose, lupus is a tuberculosis of the skin, and many forms of otorrhea are really tuberculosis of the ear. The ætiology of "scrofula" is therefore in the main identical with that of tuberculosis (*q. v.*), and this explains why the old physicians habitually insisted upon the intimate relationship between the two diseases. It was formerly thought that scrofula often terminated in tuberculosis—that is, a scrofulous child is apt to suffer eventually from tuberculosis of the lungs, intestine, or brain. To-day we know that most scrofulous children do not become, but that they are already, tuberculous.

Besides tuberculosis, hereditary syphilis and that acquired in childhood may produce symptoms that formerly were ascribed to scrofula. Syphilitic diseases of the bones, nose, skin, etc., sometimes produce clinical pictures which in all respects resemble those described above. But with our present diagnostic knowledge and technical appliances, we can generally succeed in determining the true nature of the morbid processes in question.

But there are certain other diseased conditions that can be ascribed neither to tuberculosis nor to syphilis, such as certain persistent and diversified diseases of the skin and of the mucous membrane which continually reappear, as well as certain general disturbances of nutrition all pointing to a constitutional cause.

A. Czerny has grouped all these processes under the collective name of "exudative diathesis," and has sought to substitute this conception for that of scrofula, formerly used. In these cases we have to deal with exudative processes (urticaria, eczema, impetigo, scrofula of the skin, catarrh of the mucous membranes, secondary enlargement of the lymph-glands, etc.) which seem to have grown out of a common soil. The "exudative diathesis" not infrequently appears in different members of the same family. The disorder grows with improper nourishment of the child, and is repressed by proper nourishment.

It would be difficult at the present time sharply to define and limit the idea of "exudative diathesis." We believe, however, that there are proper grounds for its assumption, and that the term "exudative diathesis" should, at least partially, replace the older one of "scrofula," which has ceased to be serviceable. What inclines us to accept the theory of such a constitutional anomaly is the fact that in adults also we observe a group of peculiar diseased conditions that might not improperly be included under the idea of "exudative diathesis." We refer to certain forms of chronic urticaria and multiple, so-called, angioneurotic, circumscribed œdema of the skin to peculiar paroxysmal disturbances of secretion of the nasal mucous membrane, to some forms of bronchial asthma, to attacks of "nervous" gastroxynsis, to the extremely remarkable clinical picture of colica mucosa, to various peculiar forms of intermittent swellings of joints and of constantly recurring and rapidly disappearing periarticular, apparently periosteal, swellings of the fingers, etc. All these diseased conditions are characterized by the fact that the same "exudations" always recur without any provocation in the persons "predisposed." In such cases we almost always have to do with so-called "nervous" persons, as in mucous colitis and nervous bronchial asthma, and with diseased processes which are usually resistant, and which, as experience teaches, can be ameliorated, if at all, far more by general measures than by local treatment. All the morbid conditions mentioned have heretofore been described as resting upon a "nervous" basis. But, in accordance with the views expressed by Czerny, it does not seem unjustifiable to us here also to speak of an "exudative diathesis." Other pediatrists (Escherisch, etc.) now lay the greatest stress upon a hyperplasia of the lymph-glands and the lymphatic organs (tonsils, follicles at the root of the tongue, follicles in the intestinal mucous membrane, thymus), and would substitute the conception of a "lymphatic diathesis" ("status lymphaticus") for the old one of "scrofula." We see here that modern medicine cannot do entirely without the old constitutional idea. A more detailed discussion of these questions would certainly lead us too far.

Treatment.—After what has been said, a comprehensive discussion of the treatment of scrofula and of the exudative diathesis is entirely out of the question. It must be again emphasized that, in the treatment of most cases of this class, the main attention is to be paid to the general hygienic and dietetic measures rather than to the special treatment of any local disorders.

According to old medical experience, cod-liver oil and salt baths have a special reputation in the treatment of "scrofula." If circumstances permit, the best way is to visit some place where there are brine baths, such as Kösen, Sulza, Salzungen, Arnstadt, Kreuznach, Münster am Stein, Rehme, Reichenhall, Ischl, or Colberg.

Treatment at these resorts is preferable to the use of artificial baths at home, because it is under more favorable hygienic surroundings. The use of sea baths (Wykauf Föhr, Rügen, and many others) is highly to be recommended in many cases.

It is therefore the duty of the physician in every case of "scrofula" to analyze the ætiology and symptoms carefully, in order to determine with what he has to deal. "Scrofula" should be regarded merely as a short way of naming a certain group of symptoms. It is convenient to retain the term as being less likely to startle the friends of the child than would the true name of the disease.

We may briefly mention a few facts with regard to the treatment of scrofulous swelling of the lymph-glands. Painting the overlying skin with tincture of iodin is a very common practice, but it seldom does much good. We have obtained more satisfactory results from iodoform ointment, or from the repeated inunction of sapo viridis. For particulars as to the opening of abscesses, or the extirpation of glands, we must refer to works on surgery.

VIII. DISEASES OF THE NERVOUS SYSTEM

I. DISEASES OF THE PERIPHERAL NERVES

SECTION I

DISEASES OF THE SENSORY NERVES

CHAPTER I

GENERAL REMARKS UPON THE DISTURBANCES OF SENSIBILITY

THE disturbances of sensibility, like all other functions of the nerves, are manifested in two directions. Under pathological conditions we observe either an abnormal diminution or a complete absence of sensibility (anæsthesia) or a morbid increase (hyperæsthesia). While in anæsthesia the ordinary or even the strongest irritations which excite the sensory nerves produce only a weak and insignificant sensation, or even no corresponding sensation at all, in hyperæsthesia very severe and painful sensations are caused by weak irritations. The "symptoms of sensory irritation" are to be distinguished from hyperæsthesia, although they are often present along with it. By this term we mean sensations which cause internal irritation, not from without, but from certain abnormal morbid conditions in the nerve itself. In the region of cutaneous sensibility, with which we shall chiefly concern ourselves in what follows, these symptoms of sensory irritation show themselves partly as actual pain and partly as the so-called paræsthesiæ—that is, abnormal sensations in the skin, which are termed "formication" (the crawling of ants), "prickling," "numbness," "a furry feeling," "hot and cold feelings," etc.

The Different Forms of Sensibility (Cutaneous and Muscular Sensibility) and the Methods of Testing Them.—As is known from physiology, the irritation of the sensory cutaneous nerves produces in us a number of sensations, differing in quality according to the manner in which the irritation acts. If, therefore, we would obtain an accurate estimate of the condition of the patient's cutaneous sensibility, we must make a special test of all the different forms of sensation; for we often see that the disturbances of sensibility do not involve all the forms mentioned alike, but that one kind of irritation is followed by perfectly normal sensation, while there is more or less complete anæsthesia for another kind. We term such partial anæsthesias of the skin, which are manifest toward only one or more forms of irritation, "dissociated" or "partial anæsthesias." The exact study of these partial anæsthesias is the more interesting, since, according to our present physiological theories (Blix, Goldscheider, etc.), the different qualities of cutaneous sensibility are transmitted to the consciousness by special nerve fibers, so that there are in the skin different terminal nervous organs for the perception of touch and superficial pressure stimuli, pain, cold and heat stimuli, etc. The fibers for the different forms of cutaneous sensibility have separate courses in the spinal cord (see the following chapter). The testing of these various forms of sensibility is, therefore, of great value in localizing morbid processes in the spinal cord. In addition to cutaneous sensibility, that of the deeper structures (fasciæ, muscles, joints) should always be tested at the same time.

The various forms of cutaneous sensibility which must be tested separately in every carefully conducted sensory examination are: touch, heat and cold, pain, pressure, and the so-called muscular sense—i. e., the sensations of position and motion.

1. *Sensibility to Contact.*—Sensations of touch are produced by simple light mechanical stimuli of the surface of the skin. There is no radical difference between "touch" and the lightest "pressure stimuli." We must take care that the deeper structures (fasciæ, muscles, etc.), are not mechanically irritated by the pressure. The superficial cutaneous sensibility is best tested by means of the hair. Every slight bending of the hair immediately produces a sensory impression, caused by the light mechanical pressure or pull on the nerve terminations which are so abundantly present in the hair follicles (M. von Frey). In the parts of the skin not supplied with hairs, the so-called Meissner's corpuscles are probably the receptive organs for contact stimuli. The well-known experience that gentle stroking of the skin is more clearly perceived than simple touch, is explained by the fact that stroking of the skin produces a stronger mechanical stimulus (lanugo hairs, folds, and wrinkles). The examination of the sensibility of the skin to simple contact is usually performed by repeatedly touching or stroking the part of the skin to be tested with the finger, a fine brush, or some other blunt object (not of metal, in order to exclude sensations of cold) while the patient's eyes are shut, and asking the patient to say whether he has felt the touch or not. Whenever hairy portions of the skin are to be tested, it is recommended to test the sensibility of the hairs (i. e., of the hair follicles). All tests of sensibility are generally conducted with the patient's eyes closed. When it is necessary to call the patient's attention to the investigation, it is always best to do so afresh by asking "Now?" and then either really to touch the skin or else to ask the question when only pretending to touch it. In this way we can best guard against errors which otherwise may readily arise from a lack of attention or of practice on the part of the patient. We may often make the patient close his eyes and then count the number of times we touch the skin, or we touch the skin in different parts of the body (alternating on the two legs, etc.), and then ask him to tell merely the place where he is touched (*vide infra*). The degree of correctness of his answer of course shows the condition of the sense of contact. The comparison of the sensibility in symmetrical parts of the body is of importance, especially in unilateral disturbances.

In a good many cases the patients feel the slightest touch, but nevertheless they say that the touch on the affected part is "more indistinct," "duller," in short, "different" from that on the normal part of the skin.

2. *Sensations of Heat and Cold* (*Temperature Sense*).—As we have already said, physiological investigations (Goldscheider, etc.) have lately led us to believe that sensations of heat and cold are to be regarded as due to two distinct functions of the cutaneous nerves. These functions are also performed by special nerve terminations and nerve fibers. If we test the sensibility of the skin for heat and cold with a pointed metal rod, heated or cooled, we can easily be convinced that the hot rod excites a sensation of warmth only on definite parts of the skin, and that the cold rod excites a sensation of cold only on other definite points ("warm points," "cold points"), but on the intervening points these rods excite no sensation of temperature. From repeated personal observation we can state that pathological symptoms agree completely with this theory of the existence of special nerve conduction for sensations of heat and cold, for we see that changes in the temperature sense in the skin by no means affect the heat sense and the cold sense equally. It is, therefore, always necessary to test both forms of the temperature sense separately. We often find that while one form of temperature sense is in normal activity, the other is much altered—a pronounced partial anæsthesia to heat or cold. The affected sense may be wholly lost or merely blunted, so that hot water is felt merely as tepid, or ice as cool. If the temperature sense be lost, the application of a hot or cold object will cause only a sensation of touch, and not of temperature. Extreme temperatures, especially extreme heat, excite, as is well known, a sensation of pain. This may, of course, remain normal; but if there is also analgesia (*vide infra*), it disappears.

If anæsthesia to cold be present, patients often speak of having a distinct sensation of warmth when the skin is touched with a bit of ice. This symptom, which we discovered and called "perverted temperature sense," may perhaps be explained by supposing that the heat nerves are excited by the severe irritation from cold.

Besides the sensations of temperature we usually test also the patient's capacity to distinguish differences of temperature. Within the moderate degrees of temperature, 80° to 100° F. (25° to 35° C.), differences of 1° F. (0.5° C.) are easily distinguishable under normal conditions, and even 0.5° F. (0.2° C.) can be distinguished on the face and fingers, but only about 2° F. (1° C.) on the back.

Variations in the temperature sense are very common. We often find them (especially in spinal disease) in those cases where the simple sensibility to touch is fully retained. The test of the sensibility to heat and cold should therefore never be omitted in testing the sensibility. It can be done with sufficient accuracy for all practical diagnostic purposes simply by touching the skin with large test-tubes filled with ice or hot water. The various thermæsthesiometers that have been devised are too complicated for practical purposes. For a hasty test it is sometimes quite serviceable to try whether the patient can distinguish warm breathing upon a given portion of the skin from cool blowing upon it.

3. *Sensation of Pain.*—The sensibility to pain, as M. von Frey has demonstrated, is probably transmitted by special nerves, the end organs of which

must be sought for in the intra-epithelial nerve terminations. For this reason sensations of pain always require stimuli which penetrate the deeper epithelial layers (pin pricks, firm pinching, strong heat stimuli). Sensations of pain are always produced by such stimuli, which are attended by a certain injury of tissues, especially of the nerve terminations, as by severe pressure ("contusion"), by strong heat stimulus ("burn"), etc. Pain has the all-important and useful task of warning us of the occurrence of an event which may produce some damage to the body, and of calling forth some defensive measure. Under pathological conditions, cutaneous sensations of touch and pain are by no means always parallel. We sometimes see that a patient does not feel a simple touch on the skin, when sticking a needle into it is immediately painful; while, on the other hand, we still more frequently find that a patient feels quite a light touch on the skin, but that the most marked irritation, such as pinching or pricking it, does not excite the slightest pain, but is felt only as a simple touch, or at most as a slight pressure. This latter condition of sensibility, the loss of cutaneous sensibility to pain with retained tactile sensibility, is termed analgesia. Both in peripheral and in central nervous diseases analgesia is a symptom that may be quite frequently observed.

The test of sensibility to pain may be best performed by pricking with the point of a pin, and also by pinching a fold of the skin with considerable force. The simple test, whether a patient is able to distinguish between the head and the point of a pin, is much employed. We thus obtain information at the same time of the sensibility to touch; for a prick with the point of a pin normally excites a slight pain, while the application of the pinhead causes a sensation of simple touch or slight pressure. In testing the sensibility to pain we must be careful to prick deep enough (penetrating the epidermis), but at first we must employ only brief pin pricks. Only when these are not felt as painful should we use long-continued pin pricks. Very often by summation of the irritation (*vide infra*) a lively sensation of pain is produced. If there be marked analgesia we can, of course, stick a pin deep into the skin or push it through a fold of skin without causing pain.

4. *Sense of Pressure.*—By pressure sense proper we mean those deep sensations of pressure which are produced by stimuli acting on the deeper structures (fasciæ, tendons, muscles, periosteum). By very simple experiments we can readily demonstrate to ourselves that these structures are individually sensitive to pressure; thus, for example, every healthy individual can clearly distinguish whether pressure is exerted down to the bone or not. Therefore, the differences in the degree of pressure are not estimated simply according to the intensity of the stimulus, but rather according to the local action (in respect to depth) of the pressure stimulus. The test of the pressure sense enables us, therefore, to judge directly of the sensitiveness of the before-mentioned deeper structures. The well-known statement of E. H. Weber that we do not estimate the intensity of the pressure sensations according to the absolute increase of the stimulus, but only according to its relative increase (about one twentieth to one thirtieth of the original strength of the stimulus), requires a different interpretation now than formerly.

According to our experience, the clinical examination of the sensitiveness to pressure of the deeper structures is of the greatest importance. It occasionally shows distinct anomalies which could not be demonstrated in any other way. Partial paralysis or diminution of the pressure sense is by no means rare in diseases of the spinal cord (tabes, myelitis, compression, tumors), and also in diseases of the brain (particularly when associated with spinal disease, infantile cerebral palsy, etc.). It is noticeable, but readily understood from the preceding explanation of the interpretation of the deep pressure sensations, that anomalies of the pressure sense are often associated with anomalies of the so-called muscular sense (*vide infra*), while, on the other hand, the pressure sense is generally normal when the muscular sense is unimpaired.

Different methods and instruments, such as von Eulenburg's "baræsthesiometer," have been devised for testing accurately the sense of pressure in patients, but they have entered but little into practice on account of their elaborate character. For the ordinary demands of practice it is sufficient to test the pressure sense by applying the finger or any blunt object. By pressing with the finger we can vary the pressure, make it weaker or stronger, and thus learn whether the patient is able correctly to state all these differences.

5. *The So-called Muscular Sense, Especially the Sensations of Motion and Position.*—A number of sensations are classed together under the names of "muscular sense" or "muscular sensibility." They are not all of the same value. Most frequently we call our power to be informed of the position of any of our limbs without the help of our eyes, and of the extent of any motion made by them, the "muscular sense." If we put a healthy person's arm into a given position when his eyes are closed, and tell him to put the other arm into the same position, he can do it with considerable accuracy. If we make passive movements in any of the joints of the extremities, a healthy person whose eyes are closed can easily and correctly state the form and direction even of the slightest of these movements. In nervous patients, however, this power may be diminished or lost, and we often speak of "disturbances of the muscular sense"; but we must note that the judgment as to the position of the limbs and the passive movements executed with them is not exclusively due to the sensibility of the muscles, but probably depends also upon the sensibility of the joint surfaces, ligaments, and tendons. All of these parts, as well as the skin, are relaxed or made tense in the different movements. Nevertheless, we believe that it is the change in muscle tension that chiefly enables us to judge of the position and movements of limbs. It would, therefore, be more correct to speak of sense of position (the sensation of position of the limbs) and sense of motion (sensation of passive movements) instead of the "muscular sense." Concerning the terminal nerve organs of the deeper structures, our knowledge is at present still defective. In the muscles the so-called "muscle spindles" are generally considered to be the terminal nerve organs.

Sensibility to movement is tested simply by making the patient close his eyes, by taking an arm or a leg firmly in both hands, and then by making passive movements up and down, right and left, at first extensive and then gradually less. The patient should state the direction of the movement made. We may also describe with the extremities different letters or figures in the air, which the patient should recognize with his eyes closed. If the test is to be absolutely accurate, passive movements must be tested in all the joints individually (shoulder, elbow, hip, knee, etc.).

In important cases one must never neglect to test accurately the sensations of motion in the distal joints (hand, finger, foot, toes), since frequently marked disturbances are found here when the sensibility to movement of the large proximal joints (shoulder, hip) is still almost completely unimpaired.

The sensibility for the position and posture of the limbs is tested by putting one extremity passively into a given position and then asking the patient to put the other corresponding extremity into the same position, as nearly as possible. It is still better to have the patient make some definite movement, especially to point to or seize some object, at first with the eyes open, then to let him close his eyes and repeat the movement as well as he can.

Marked disturbances of the sensation of position and motion are found most frequently in tabes dorsalis (*vide infra*), but not infrequently, too, in other diseases of the spinal cord, in cerebral hemianæsthesia and hemiataxia, as well as in hysterical anæsthesia and paralysis.

The sensation of the strength of voluntary muscular contraction, the so-called "sense of effort," is quite different from the sensations just described. Besides the peripheral sensations in the muscles and tendons we perhaps have to do with special sensations of innervation. In raising weights we can distinguish with considerable accuracy the lighter from the heavier. In such cases, also, we deal not with the absolute, but with the relative, differences in weight; we can usually tell quite plainly when one fortieth of the original weight has been added or taken away. The sense of effort, then, is somewhat finer than the sense of pressure. In order to exclude the latter in the test we have the patient lift the weight, suspended in a towel, with his hand or foot, but in the lower extremities it is scarcely possible to exclude entirely the co-existing sensations of pressure.

We must mention, in conclusion, that muscular contraction is in itself accompanied by a sensation, as may be proved, for example, in irritating the muscles by faradism—electro-muscular sensibility. In certain forms of spasm the muscular contraction becomes so strong that it causes a decided pain, which is probably due to irritation of the sensory muscular nerves discovered by C. Sachs.

Tests of the sense of effort and of the electro-muscular sensibility have thus far been but little employed in nervous diseases. They also have no great practical significance.

6. *Electro-cutaneous Sensibility. Vibration Sensibility.*—The test of cutaneous sensibility by means of the electric current has been proposed by various observers. The advantage of it is that in this way the intensity of the irritation can be very easily and accurately graded and expressed in numbers, by the position of the coil in using the faradic current, or by the galvanometer in using the constant current. The faradic current is usually employed in testing the sensibility, and we designate it by the position of the coil when the first sensation is felt and the position when the first pain is felt. Pathological deviations are given by comparison with normal portions of the skin (testing, if possible, symmetrical parts) or with healthy people. The galvanic current is also used to test sensibility, noting the occurrence of a burning sensation at the cathode. For practical purposes, the test of electro-cutaneous sensibility is unnecessary, since its results are the same as in testing the sensibility to touch and to pain. The testing of vibration sen-

sibility [osseous sensibility] by placing tuning forks on the bones and soft parts is equally unnecessary.

7. *The Localization of Cutaneous Impressions. Sense of Locality.*—Under normal conditions, as we know, not only do we feel the touch of an object, but we can tell with a good deal of accuracy the place on our skin which was touched. This power we term the ability to localize our sensation. In nervous patients we often see that, while cutaneous sensibility is still present (we refer not only to tactile sensibility but also to the other forms), it is localized more poorly and with less accuracy than is the case under normal conditions.

In the simple test of the sense of contact we may also examine, at least roughly, the power of localization if we ask the patient also to state where the touch is felt, or if we ask him to designate with his hand as accurately as possible the part of the skin touched. A more accurate method was proposed by E. H. Weber. It consists in determining the smallest distance which must separate two simultaneous cutaneous irritants from each other, in order that they may be perceived as two locally distinct sensations. Weber has found that this distance differs very much in different parts of the body, and from this he has divided the whole surface of the skin into so-called tactile circles. As data for the examination of patients, some of the figures obtained by Weber in healthy individuals may here be given: The smallest distance at which the two points of a pair of compasses[1] applied at the same time to the skin may be plainly distinguished from each other is 11 to 15 mm. on the cheeks, 6 mm. at the tip of the nose, 22 mm. on the forehead, 1.2 mm. at the tip of the tongue, 4 to 5 mm. at the back of the tongue and on the lips, 34 mm. on the neck, 77 mm. on the upper arm, 40 mm. on the forearm, 31 mm. on the backs of the hands, 11 to 16 mm. on the backs of the fingers, 2 to 3 mm. at the tips of the fingers, 55 to 77 mm. on the back, 45 mm. on the chest, 77 mm. on the thigh, 40 mm. on the leg, 40 mm. on the instep; but these figures show certain variations in different individuals, so that they are to be regarded as only average values.

Testing the sense of locality by Weber's method has no great practical importance; it also takes very much time and demands intelligence, patience, and good will on the part of the patient. The influence of practice is manifested in a very remarkable way, since the perceptible difference becomes considerably less if the examinations are often repeated. On the other hand, a single examination, as in testing any form of sensibility, must not be too long protracted, for otherwise the patient may easily become fatigued, and the data obtained will be entirely contradictory. If we test the sense of locality by bringing down the two points not at the same time but one after the other, and vary it by touching the same place twice, or a different place each time, we obtain from the outset, as we have repeatedly proved, smaller numbers than if we touch the skin with the two points of the compasses at the same time. We also obtain somewhat different values for the fineness of the sense of locality if we test the so-called sensations of motion (Leube)—that is, the distinction between a simple circumscribed touch of the skin and a very short line drawn with a stick on the skin. In this way we can also determine whether the patient can distinguish accurately the direction of transverse and longitudinal lines.

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[1] There are special "tactile compasses" with blunt ivory points and graduated quadrants.

We may also mention here two peculiar anomalies of sensibility, *polyæsthesia* (G. Fischer) and *allochiria* (Obersteiner). Polyæsthesia is a symptom shown by certain patients (especially patients with tabes), who, when the skin is touched with only one point of the compasses, have a sensation as if they felt two or even more points. The cause of this remarkable anomaly of sensation is not yet adequately explained. In allochiria a stimulation of the skin (touch, pain) is felt, not on the part touched, but on the corresponding point on the other half of the body. This remarkable symptom has been observed a few times in cerebral hemiplegia, etc.

8. *Delay of Sensation (so-called Delayed Conduction), Summation of Irritation and After-sensations.*—Quite frequently in diseases of the spinal cord, especially in compression paralysis and tabes (*q. v.*), and not infrequently in peripheral-nerve disease (neuritis), we see a marked delay in sensation after the action of an irritant. This symptom affects chiefly the sensibility to pain. If in such a case we stick a pin into the sole of a patient's foot, several seconds elapse, even ten or twenty, it is said, before the pain is felt. As was first observed by Naunyn and E. Remak in tabes, and as has often been confirmed since, after sticking a pin into the foot there is in such cases first a simple sensation of slight pressure, and some seconds later the peculiar sensation of pain, so that the patient at once responds to the prick with "Now," and a little later with "Ow!" as an expression of pain.

On analyzing this symptom somewhat more closely we will have to distinguish between single brief and continuous irritations (pin pricks). If we make, for example, a single short prick in the skin, it may happen that the pain thus caused appears comparatively late. This symptom depends perhaps upon a "delay of conduction," although it is still undetermined, of course, whether this delay takes place actually in the peripheral-nerve fiber or in the interpolated ganglion cells. The conditions in permanent painful excitation (continued pin prick) are different. In these cases we notice very often that immediately after the insertion of the pin the patient feels nothing or only a simple touch. If the prick be continued, however, there is felt a lively sensation of pain, often only after a few seconds. This phenomenon is apparently due, not to a delay of conduction, but to a summation of the irritant action, which thus gradually attains sufficient intensity finally to break through the existing resistance and to reach the consciousness in full strength. At the same time with the sensation of pain there is usually a reflex twitching, and the same conditions are therefore to be considered in testing the cutaneous reflexes (*vide infra*).

In this connection, it is well to mention briefly the after-sensations, which we have noted in the most striking fashion, especially in cases of tabes. After a single short prick these after-sensations appear as a burning feeling in the skin, which lasts a strikingly long time, or else the first sensation soon ceases after the prick, and then, in the same spot on the skin, new sudden sensations of pain are felt several times, just as if the patient had been pricked anew.

9. *The Special Sensibility to Touch (Stereognostic Sense).*—The highest and most valuable function of the skin as a sensory organ is the judgment we can make of the size, form, hardness, and consistency of objects by feeling

of them—in short, the recognition of them. This function, the special sense of touch, is apparently a complicated estimate of all the separate sensations derived from the different cutaneous nerves, to which are always added at the same time a large number of sensations of movement and sensations of position. For "touch" and "feeling" always imply a movement of the parts which touch. For this reason, in man, the function of "touch" is chiefly localized in the highly sensitive and extremely mobile fingers. We see, therefore, that a patient whose hand is completely paralyzed can recognize objects either with difficulty or not at all by the touch, although the cutaneous sensibility is retained. If there be actual anæsthesia of the skin it is, of course, also impossible to recognize objects by the sense of touch, although mobility is retained. We estimate the size and shape of objects principally by our muscle sensations, its consistency and surface by means of the pressure sense, temperature sensations, etc. There are, however, also cases where the hand can be moved well enough and where the sensibility of the skin and muscles for the different qualities of sensation is retained, but nevertheless the recognition of objects by the sense of touch is markedly impaired. In such cases there is probably always some cerebral (cortical) disturbance, which may be regarded as analogous to cortical blindness and cortical deafness—that is, the normal mental estimate and consequently the "understanding" of the various cutaneous sensations received. The author has seen such conditions, especially in cerebral infantile paralysis; the intelligent child feels every touch on the skin perfectly well, but, when his eyes are closed, he cannot recognize by feeling of them with the affected hand the most familiar objects (key, knife, ring, money, etc.). At any rate it is very important, especially in cerebral diseases, not to neglect this mode of investigating the special "sense of touch." If we choose for our tests simple stereometric figures (sphere, cube, cone, octahedron), we may also speak of a "stereognostic sense." In every case of disturbance of the stereognostic sense, all the different forms of cutaneous and especially muscular sensibility (sense of position, deep-pressure sensibility) must be examined minutely, in order to decide whether the faulty recognition of the object felt is due to lack of sensibility or to inability to interpret the sensory impressions.

CHAPTER II

THE SENSORY CONDUCTION TRACTS AND ANÆSTHESIA OF THE SKIN

Course of the Sensory Conduction Tracts.—In the previous chapter we discussed the different varieties of cutaneous and muscular sensibility; we will now take up the question of the paths by which these sensory stimuli are conducted from the periphery to the central organ. In the first place, we must consider the origin of the peripheral sensory nerves; for the peripheral sensory nerve, with its terminal organs in the skin for the reception of external impressions, must be regarded in the same way as every other nerve fiber, viz., as the direct prolongation of a nerve cell (ganglion cell). We now know definitely that all peripheral sensory nerve fibers come from the cells of the spinal ganglia (intervertebral ganglia). All, or at least most of these cells, have a short process which soon divides into two offshoots from the cell; a long one, going to the surface of the body (the peripheral sensory nerve), and a shorter one, which enters the posterior root of the spinal cord, and is intended for the further conduction of sensory impressions. Almost all the fibers of the posterior roots accordingly come from the spinal ganglia; during fœtal development they grow from their cells of origin into the spinal cord. The spinal ganglion cell with its two processes, the one going to the periphery of the body (peripheral sensory nerve), the other to the spinal cord (posterior root fiber), forms the first (peripheral) sensory neuron. All stimuli originating from the surface of the body and the deeper structures (muscles, etc.), in the first place, reach the spinal cord by means of the posterior root fibers. The sensory cranial nerves (trigeminus, vagus) must be considered as absolutely homologous with the sensory spinal nerves.

As soon as the posterior roots have entered the spinal cord, a very marked separation of the fibers occurs (see Figs. 114 and 115); one (median) part enters the white posterior column of the cord, while another part continues into the gray posterior horn. A third part (Fig. 115, 4) goes to the anterior horn and breaks up into arborizations around the motor cells—these are evidently paths for reflex movements.

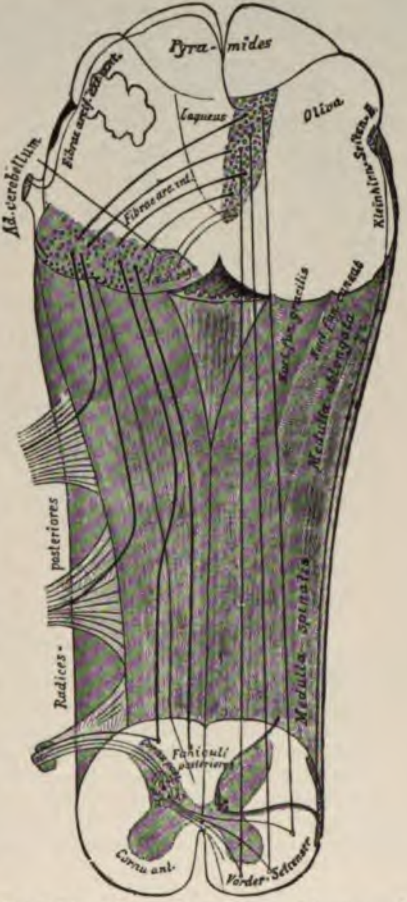

FIG. 114.—(From EDINGER.) Diagram of the course of the posterior sensory root fibers in the spinal cord and medulla oblongata. *Vorder-Seitenstr.* = Anterolateral columns. *Kleinhirn-Seiten. B.* = Lateral cerebellar tract.

Let us first consider the fibers entering the posterior columns. Soon after their entrance the fibers divide again into a short descending and a long ascending branch. The short descending branches serve, as I suspect, for reflex processes (*vide infra*), while the ascending fibers in the posterior columns form the special continuation of the centripetal tract. That portion of the posterior columns into which these root fibers enter is called the "posterior root zone," or "root entrance zone." Since new root fibers are constantly entering the cord as we go upward, the fibers coming from the lower portions, and especially from the lumbar cord, are gradually pressed toward the median line by the new fibers that enter. Thus it happens that the fibers from the lumbar and the lower dorsal cords are in quite a median position in the cervical cord, in the region of the so-called columns of Goll, while the fibers arising from the posterior roots of the cervical nerves pass upward in the column of Burdach. All these fibers which extend upward in the posterior columns end in the nuclei of Goll's and Burdach's columns in the beginning of the medulla oblongata. From the cells of these nuclei come the secondary sensory neurons, whose fibers decussate in the medulla oblongata (fibræ arcuatæ internæ, or sensory decussation in the lemniscus, Fig. 114), and in their further course form the so-called lemniscus, which passes through the medulla, pons, and tegmentum of the crus to the cerebrum (see Fig. 116).

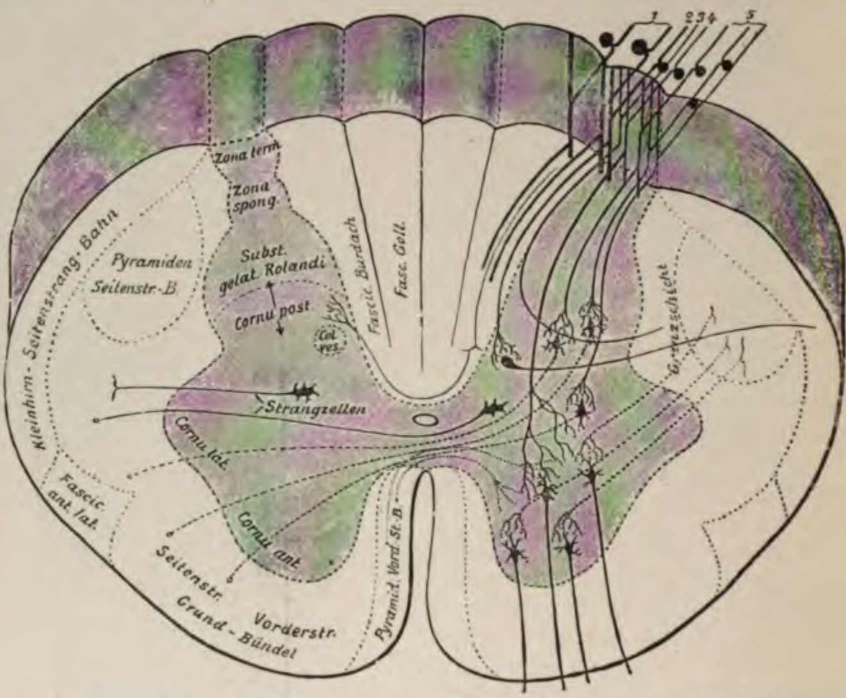

FIG. 115.—(From EDINGER.) Diagram of a transverse section of the spinal cord with especial regard to the course of the posterior root fibers: 1. Fibers in the posterior column. 2. Fibers to the cells of Clarke's column, whose processes enter the lateral cerebellar tract. 3. Direct fibers in the "lateral limiting layer" of the lateral column (not mentioned in the text). 4. Fibers to the motor cells of the anterior horn (reflex fibers). 5. Fibers in the posterior horn splitting up about its cells. *Bahn*, tract; *Grenzschicht*, limiting layer; *Grund-Bündel*, ground bundle; *Kleinhirn*, cerebellum; *Pyramiden*, pyramid; *Seitenstrang*, lateral column; *Vorderstrang*, anterior column; *Strangzellen*, cells to columns.

A second (lateral) portion of the fibers of the posterior roots enter at once the gray matter of the posterior horn. These fibers also divide into an ascending and a descending branch. A number of especially fine fibers remain in the periphery of the posterior horn (*zona terminalis*, Fig. 114, also called Lissauer's zone). The fine fibers probably enter the posterior horn through the zona spongiosa, and enter into similar relations with the ganglion cells as the other coarser fibers of this portion of the posterior root fibers, which enter the posterior horns directly through the substantia gelatinosa, and after a short course split up in so-called terminal arborization about the ganglion cells there. With these cells begin the second sensory neurons for this part of the sensory tract; the processes (fibers) of these cells at once enter the lateral column of the opposite side through the anterior commissure, decussate therefore in the anterior commissure of the cord, ascend in the anterior and middle portions of the anterolateral columns (anterior and internal to the motor pyramidal lateral tract), reach the medulla oblongata also in the lemniscus, and unite here with the above-described fibers from the cells of Goll's and Burdach's nuclei. The accompanying diagram (Fig. 116, the red portion *L*, signifying the lemniscus) gives an idea of the position of the lemniscus—that is, of the chief sensory tract—farther upward in the pons and tegmentum. Higher still, in the internal capsule, the sensory tract lies in the posterior limb behind the motor pyramidal tract (*vide infra*, Fig. 125). There is still considerable obscurity as to its further course. A part of the

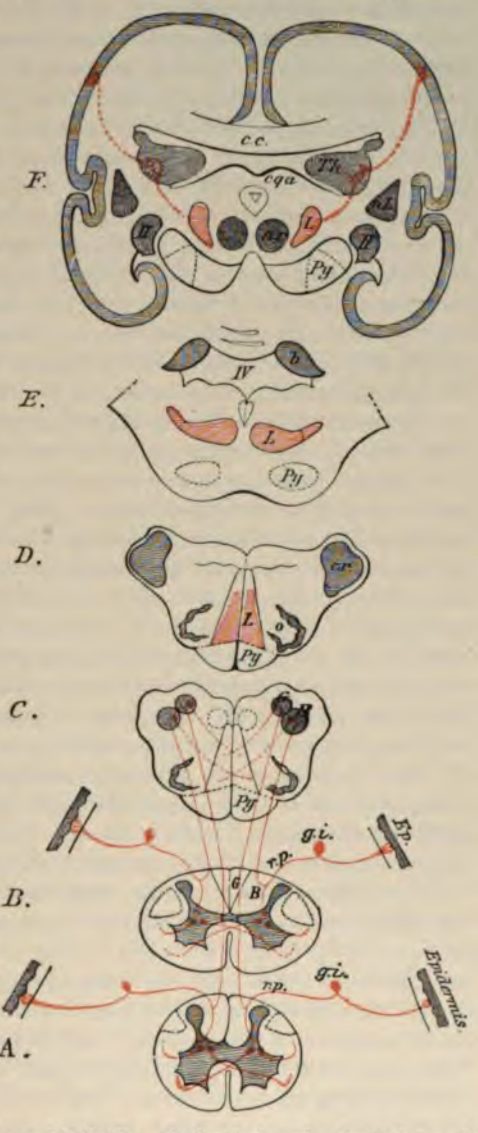

Fig. 116.—Diagram of the course of the sensory conduction tracts: *A*. Entrance of the posterior sensory root fibers in the lumbar cord. *gi*. Intervertebral ganglion. *rp*. Posterior root. A part of the fibers that enter end in the posterior horns, from whose cells new fibers arise and enter the lateral columns, partly crossed, partly uncrossed. Another part of the fibers from the posterior roots pass upward in the posterior columns and form in *B* the so-called columns of Goll (*G*). *B*. Cervical cord. Lettering as in *A*. *B*. Columns of Burdach. *C*. Medulla oblongata. Region of the decussation of the lemniscus. The fibers from the posterior columns end in the nuclei of Goll's and Burdach's columns (*G* and *B*). New fibers arise from the cells of these nuclei, which form the decussation of the lemniscus. *Py*. Motor pyramids. *D*. *L*. Sensory lemniscus. *ol*. Olivary body; *cr*, corpus restiforme. *E*. Section through the pons. *L*. Lemniscus. *b*. Superior cerebellar peduncle. IV. Fourth ventricle. *F*. Frontal section through the region of the corpora quadrigemina. *L*. Lemniscus. *nr*. Red nucleus. *Py*. Pyramidal tract in the crusta of the crus cerebri. *nL*. Lenticular nucleus *Th*. Optic thalamus (interruption of the sensory tract? Beginning of a third sensory neuron?). *cqa*. Anterior corpora quadrigemina: II. Corpus geniculatum externum and optic nerve. *cc*. Corpus callosum.

lemniscus fibers probably end in the ventral segment of the optic thalamus, from whose cells a third sensory neuron arises, sending its fibers to the cerebral cortex. Another part of the lemniscus fibers, however, pass directly to the cerebral cortex without interruption. The sensory fibers from the posterior columns probably end, in part, at least, in the cerebral cortex in the central convolutions, especially in the posterior central convolution and in the neighboring portion of the parietal lobe, and perhaps also of the frontal lobe. It is extremely interesting physiologically that the central terminal region of the sensory fibers in part coincides in its localization with the motor centers (*vide infra*). [The investigations of Sherrington, Campbell, and others would indicate that the sensory and motor centers do not coincide, but that the sensory centers are situated in the posterior central convolution, behind the fissure of Rolando, and the motor centers in the anterior central convolution, in front of the fissure. Horsley, however, still believes that the centers do coincide; that there is motor representation behind the fissure of Rolando and sensory representation in front of it.—K.]

The separation of the centripetal posterior root fibers, which we have just described, into two main groups—the one to the posterior columns and the other to the posterior roots—indicates, without doubt, separate physiological functions of these two fiber tracts. The clinical observations of sensory disturbances in pathological conditions of the spinal cord, especially in syringomyelia and the so-called unilateral lesions, permit us to draw some absolute conclusions in this respect. We can say positively that the fibers which enter the posterior horns, and soon after decussate in the spinal cord itself, are intended to convey sensations of temperature and pain, while those fibers which ascend in the posterior columns without decussating convey the muscle sensations (sensations of position and motion), sensations of deep pressure, and also sensations of simple contact. In regard to the latter, the statements of different authors are still contradictory. According to my own observations, however, it is most probable that the sensations of contact are also conveyed by the fibers of the posterior columns. It is possible, perhaps, that simple sensations of contact may travel a number of ways.

This differentiation of the conduction tracts, according to the variety of the sensation, is readily understood on a physiological basis. Sensations of pain and temperature serve less for the recognition of the external world than for reflex and regulatory purposes. Their conduction tends therefore to reach the reflex mechanism in the gray matter as soon as possible. Sensations of contact, pressure, and position together constitute the tactile sense proper. They serve us for the recognition of the external world, and for the regulation of our voluntary movements. Their conduction paths therefore ascend to the higher centers, to the brain cortex. Accurate examination of all forms of sensation is of the greatest importance in localizing lesions of the spinal cord. Diseases of the white posterior columns produce entirely different disturbances of sensibility from those found in lesions of the gray posterior horns. We differentiate, therefore, in sensory disturbances of the spinal cord, between a posterior column type (diminished sensation of contact, pressure, and motion, with pain and temperature sense preserved) and a posterior horn type (disturbance of pain and temperature sense, with preserved sensibility to contact, pressure, and motion). Naturally, a combination of the two types occurs very frequently.

[Head, Sherren, and Thompson, as a result of their researches, are doubtful as to the existence of special points in the skin for sensations of contact, pain, and temperature. They think that there are three afferent systems in the peripheral nerves. The first system conveys deep sensory impulses, sensations of pressure, the localization of pressure, pain on pressure, and the extent and direction of movements. These sensations are conveyed by fibers which run with the muscular nerves, and are preserved after division of the sensory nerves to the skin. The second system, which they term the protopathic system, supplies the whole body and conveys, through the cutaneous nerves, sensations of pain and of extreme degrees of heat and cold (temperatures above 113° F. [45° C.], or below 68° F. [20° C.]). The third, epicritic, system endows the surface of the body with sensibility to slight stimuli, a light touch, cutaneous localization, discrimination between one or two points of the compass æsthesiometer, discrimination of differences in size and intermediate degrees of temperature, 77° to 104° F. (25° to 40° C.). The fibers of this system are more easily injured and regenerate more slowly than those of the protopathic system. In the spinal cord those afferent impulses form new combinations. All sensations of pain travel along the same path in the cord, whether the pain be due to a pin prick or to pressure, and all sensations of heat and cold have their individual paths, as do sensations of motion. Sensations of touch, pressure, and localization probably follow the same tract in the cord. Most of these tracts for the conduction of the different forms of sensation cross over to the opposite side. The impulses for pain, heat, and cold begin to enter the posterior gray horn of the same side, and cross rather rapidly through the gray matter, crossing completely in the space of six segments of the cord, and ascending in Gower's tract. Sensations of touch and pressure ascend in the posterior columns for a number of segments and then cross through the gray matter to the anterior columns of the opposite side. Muscular sensibility is conveyed to the cerebellum by the cerebellar tract on the same side and to the cerebrum by the posterior column to the nuclei in the medulla of the same side, and then crosses by the median lemniscus. The ability to discriminate between two points is conveyed by fibers which pass up to the nuclei in the medulla on the same side. There are, in addition, secondary paths, by short chains in the gray matter, which also convey sensations of pain.—K.]

The General Causes of Cutaneous Anæsthesia.—Regarding the separate causes of anæsthesia, we see, in the first place, peripheral anæsthesia under conditions where the terminal organs of the sensory cutaneous nerves have lost their direct irritability. After chilling the skin, after the local action of ether and similar substances, after the corrosive action of acids and alkalies, carbolic acid, etc., as well as after the use of certain narcotics (such as cocain), we see an anæsthesia of the skin, which is due to injury of the terminal sensory organs. We may probably put the frequent anæsthesia of washerwomen in this class, for their hands and forearms are exposed all day to the action of cold, lye, etc. The anæsthesias which develop in circulatory disturbances of the skin also have the same peripheral origin, especially the "spastic anæmia," which sometimes comes in the hands and is due to a spasm of the small arteries.

We distinguish the peripheral anæsthesia of conduction, which may be produced by all forms of lesion of the nerve trunks, from the peripheral anæsthesias in the strict sense of the term. Traumatic influences, compression from new growths, and inflammation and degeneration of the peripheral nerves, as in neuritis, are the most frequent causes of this form of anæsthesia, which is often limited to the region of distribution of one or more individual nerves. The circumscribed anæsthesias which often come on after acute diseases (typhoid fever, diphtheria), and in some chronic diseases (diabetes), are usually peripheral (neuritic) anæsthesias.

Spinal anæsthesia is very often seen in the different diseases of the spinal cord, most frequently in tabes, because this, as we shall see later, is a direct disease of the peripheral sensory neuron; but spinal anæsthesia is not infrequent in diffuse acute and chronic inflammation of the cord and in compression and traumatic destruction, in new growths, and especially in syringomyelia.

Cerebral anæsthesia is seen especially in hemorrhages, foci of softening, and tumors, which affect any part of the lemniscus tract or the posterior portion of the internal capsule. Such anæsthesias affect the half of the body opposite to the lesion, and are called hemianæsthesia. As a rule, experience shows that cerebral anæsthesia of organic origin is seldom very intense. Hysterical anæsthesias, however, may attain an extreme degree; they are due to a disturbance of the perception of sensory impressions by the consciousness.

The Symptoms of Cutaneous Anæsthesia.—In many cases the patient himself notices the existence of anæsthesia. He finds that in certain parts of the body he no longer feels the pressure of his clothing or the bedclothes in the usual way. Anæsthesia of the hands is soonest noticed, because this affects the patient's occupation in diverse ways, so that, for example, he is apt to let fine objects, such as needles, drop from his hands. In other cases, of course, the anæsthesia is first found on a physical examination. It is worthy of note that hysterical anæsthesia especially, even if it be very marked and extensive, may often be wholly overlooked by the patients themselves.

Anæsthesia is very often combined with abnormal subjective sensations, paræsthesiæ, in the affected portions of the skin. The patient has a feeling there of "numbness," or a "furry feeling," or complains of prickling or formication. The anæsthetic parts may even be the seat of very decided pain (*anæsthesia dolorosa*), if there is abnormal irritation of the sensory nerves from the break in the conduction toward the center. The most diverse forms of anomalies of motility and of the reflexes, and vasomotor disturbances, may of course be present in addition to the anæsthesia. Special mention must be made of the so-called trophic disturbances (inflammation, ulceration, bedsores, etc.) which are often seen in anæsthetic regions. Whether in these cases we actually have to do in part with direct injury of the tissues from the failure of normal "trophoneurotic" influences is at present wholly undetermined. The majority of the ordinary so-called "trophic" disturbances are, without doubt, due to external injuries (slight wounds with consequent purulent infection). When there is anæsthesia, or especially analgesia, of the patient's skin, these injuries do not receive sufficient attention, and therefore they are neither avoided nor thoroughly treated (compare especially the chapter on syringomyelia). Greater external injuries—burns, bedsores, etc.—are often not noticed at all when on anæsthetic parts, and therefore they may attain an extraordinary size and become very bad.

Voluntary movements, as such, are not markedly impaired by anæsthesia of the skin, but considerable difficulty is experienced in performing all finer movements. Thus patients with diminished sensibility in the fingers can usually no longer perform any work requiring manual dexterity (e. g., fine sewing), because they lose the needle every minute. Marked organic disturbances of the deeper sensibility (muscle sensibility) probably always cause an uncertainty of movements, which is known as ataxia (*q. v.*). This motor disturbance is absent only in hysterical anæsthesia. The loss of the regulatory muscle sensations can be replaced in part by the control of the eye. With the eyes shut, therefore, the motions of the anæsthetic parts become very uncertain, both in anæsthesia of the skin and of the deeper parts, the muscles and joints, since then the patient loses entirely his power of judging of the extent and of the precise direction of his movements. Very extensive

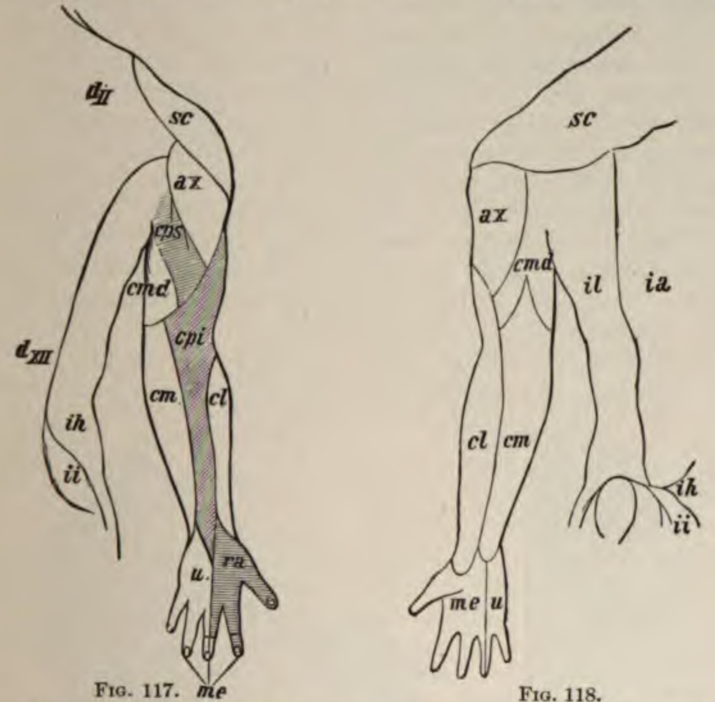

FIG. 117. FIG. 118.

FIGS. 117 and 118 [1].—Distribution of the sensory cutaneous nerves in the trunk and upper extremities. FIG. 117. Posterior aspect. FIG. 118. Anterior aspect. The shaded portion in Fig. 117 designates the territory supplied by the radial nerve. (From HENLE.) *sc.* Supraclavicular nerves (from the cervical plexus). *ax.* Cutaneous branch of the axillary nerve. *cps* and *cpi*. Superior and inferior posterior cutaneous nerves from the radial (*ra*). *cmd*, *cm*, and *cl*. Median cutaneous, median, and lateral nerves. *me.* Median nerve. *u.* Ulnar nerve. *d*II. Second dorsal nerve. *d*XII. Twelfth dorsal nerve. *ih.* Ilio-hypogastric nerve. *ii.* Ilio-inguinal nerve. *il.* Lateral perforating branches, and *ia*, anterior perforating branches of the intercostal nerves.

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[1] [Henle's nomenclature of the peripheral nerves has been adopted.—TRANS.]

anæsthesia of the skin, associated at the same time with anæsthesia of the organs of special sense, is sometimes not without influence upon consciousness. We have had under observation a very remarkable case of total anæsthesia of the whole body associated with unilateral blindness and deafness. If we entirely excluded this patient from all external impressions of sense by closing his still serviceable eye and ear, we could at once in this way put him into a deep sleep. Similar observations have been reported by other physicians. It must, of course, be said in this connection that such extensive anæsthesias have thus far been observed almost exclusively in hysteria (*q. v.*), and that, therefore, psychical factors have perhaps been also of influence in the phenomenon just mentioned.

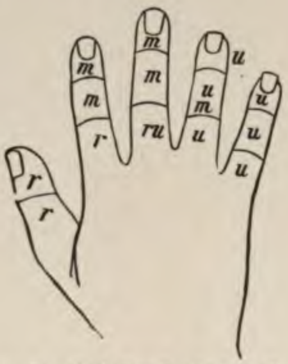

Fig. 119.—Detailed distribution of the nerves to the dorsal surface of the fingers. (From Krause.) *r.* Radial nerve. *m.* Median nerve. *u.* Ulnar nerve.

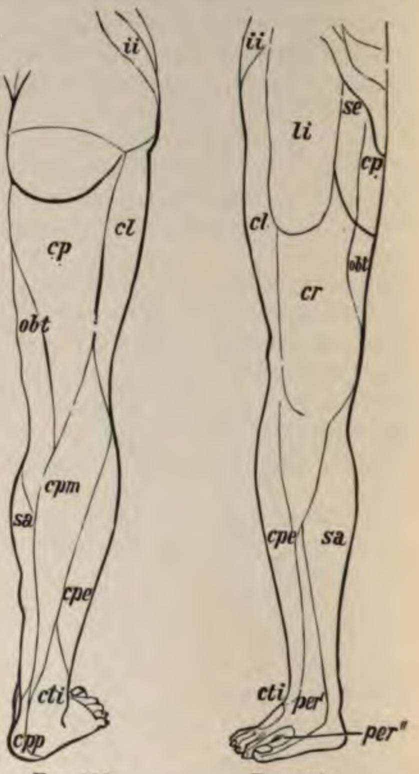

Fig. 120. Fig. 121.

Figs. 120 and 121.—Distribution of the sensory cutaneous nerves to the lower extremities. Fig. 120. Posterior aspect. Fig. 121. Anterior aspect. (From Henle.) *ii.* Ilio-inguinal nerve. *li.* Lumbo-inguinal nerve. *se.* External spermatic. *cp.* Posterior cutaneous. *cl.* Lateral cutaneous. *cr.* Crural. *obt.* Obturator. *sa.* Saphenous. *cpe.* Peroneal communicating nerve. *cti.* Tibial communicating nerve. *per'.* Superficial branch of the peroneal nerve. *per".* Deep peroneal nerve. *cpm.* Posterior median cutaneous nerve. *cpp.* Cutaneous plantar nerve.

We cannot here go into details of the different forms and varieties in the course of anæsthesia, since they will be spoken of under the different diseases which lie at the basis of the anæsthesia. The regions of distribution of the peripheral sensory nerves of the skin are given diagrammatically in the accompanying cuts (Figs. 117 to 121). Experience shows, however, that complete section of a peripheral sensory (or mixed) nerve almost never causes total anæsthesia in the corresponding cutaneous region, probably because there are many anastomoses of the terminal branches of the sensory nerves.

The cutaneous areas of distribution of the nerves from the different sensory spinal roots will be discussed later on under the pathology of the spinal cord.

Anæsthesia in the region of the trigeminus demands a brief special description.

Anæsthesia of the Trigeminus.—This is observed in tumors, syphilitic new growths, chronic inflammations, and analogous processes at the base of the skull, which compress the trunk, the Gasserian ganglion, or one of the three branches of the trigeminus, or directly involve the nerves. Traumatic lesions of the trigeminus also are not very uncommon. The distribution of the anæsthesia, according as the affection involves the whole trigeminus, or only one branch of it, may be seen in Figs. 122 and 123, but individual variations occur. In total anæsthesia of the trigeminus, the conjunctiva and cornea, the mucous membrane of the nose, the cavity of the mouth, the tongue up to the foramen cæcum, and the soft palate, are anæsthetic on the affected side. Hence we often find ulcers on the tongue and the mucous membrane, which come from being bitten. The "neuroparalytic ophthalmia," not infrequently seen in anæsthesia of the trigeminus, is of special interest, and it has been much studied by physicians and physiologists. This is an ulcerative keratitis, almost always beginning in the lower segment of the cornea, and sometimes passing over into a purulent inflammation of the whole eyeball. This affection is regarded in many quarters as an immediate result of the disturbance of special "trophic" functions, but, after careful experiments, it seems certain that external traumatic influences always occasion the first trouble, and render the intrusion of inflammatory agents possible. As a result of the anæsthesia of the eye, the patient does not notice the inflammatory symptoms, which therefore often attain an extremely high degree. It is still uncertain,

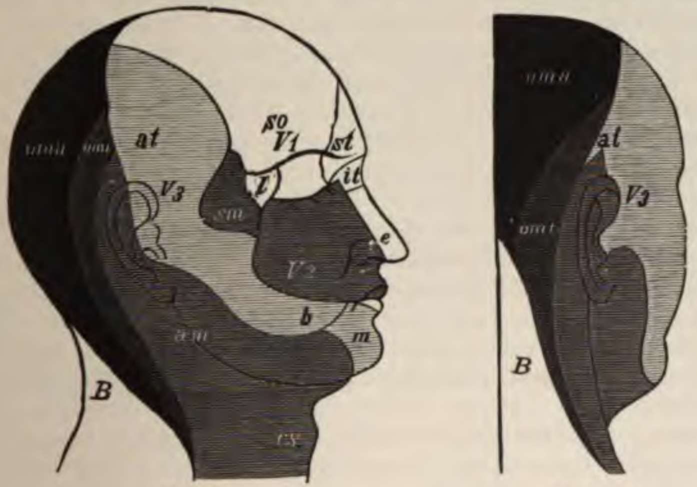

Fig. 122. Fig. 123.

Figs. 122 and 123.—Distribution of the sensory cutaneous nerves in the head. *oma* and *omi*. Occipitalis major and minor. *am*. Auricularis magnus. *cs*. Superficial cervical. V^1. V^2. V^3. First, second, and third branches of the fifth (*V*). *so*. Supraorbital. *st*. Supratrochlear. *it*. Infratrochlear. *e*. Ethmoidal. *l*. Lachrymal. *sm*. Subcutaneous malæ, or zygomatic. *at*. Auriculo-temporal. *b*. Buccinator. *m*. Mental. *B*. Posterior branches of the third cervical.

but not wholly improbable, that we must also assume a specially diminished power of resistance on the part of the tissues and a limitation of all compensating functions as a result of the nervous lesion.

The skin of the face is often somewhat bloated and reddened in anæsthesia of the trigeminus, and it is sometimes warmer, but often colder, than normal. The reflexes are lost in peripheral anæsthesia, and the lachrymal secretion ceases, or at least is persistently diminished. The taste is almost always decidedly diminished on the anterior two thirds of the tongue on the affected side, the territory supplied by the lingual nerve. According to the investigations of Krause on patients in whom the Gasserian ganglion has been extirpated (*vide infra*), the trigeminus contains fibers which conduct sensations of taste for sweet, sour, and salt from the tip of the tongue and the anterior two thirds of its lateral border, but individual variations seem to occur. The secretion of saliva, as a rule, suffers no perceptible change, but we sometimes see a diminished secretion of mucus in the nasal cavity on the affected side.

Besides the peripheral anæsthesias just described, other anæsthesias occur, especially in syringomyelia, and more rarely in other diseases of the medulla oblongata, which are due to a lesion in the region of the trigeminal nucleus (nuclear anæsthesia). In those cases a remarkable condition is manifest—namely, that, in diseases which extend from below upward, the anæsthesia first appears in the upper part of the forehead next to the hairy scalp (and usually on the lateral portion before the median), and that the sensory disturbance extends in a curve downward and inward to the eyebrows, then to the outer portion of the eyelids, then to the median portion of the lids, and finally to the skin of the alæ nasi and the bridge of the nose. The frontal portion of the first branch of the trigeminus, therefore, seems to derive its fibers from the most distal (lowest) nuclear groups, next above come the nuclei for the third branch (temporal region), and then for the second branch and the nasal portion of the first branch (see the anatomical description in the section on diseases of the medulla oblongata). The disturbances of sensibility in the mucous membrane of the mouth are usually seen first in the posterior portion of the mucous membrane of the cheeks, then in the hard palate, and finally in the region of the soft palate and the tonsils. The mucous membrane of the oral cavity, therefore, derives its sensory fibers from the distal portion of the trigeminus nuclei (Schlesinger).

Treatment of Cutaneous Anæsthesia.—Since anæsthesia in most cases is only a symptom, treatment, of course, must always first be directed against the underlying disease. Therefore, we will mention here only those measures which are to be used symptomatically against anæsthesia.

The chief remedy is the electric current. We treat the anæsthetic part of the skin with the faradic current, using ordinary electrodes, or, better still, the wire brush, or with the galvanic current, stroking the skin slowly back and forth with the cathode for two or three minutes. Sometimes we can see a result immediately after the sitting. Hysterical anæsthesia may often be removed in this way in a very short time.

Besides electricity, we prescribe embrocations, such as spirit of camphor, formic acid, spirit of thyme, etc., and also baths, and cold or hot local douches, combined with rubbing the skin. The action of internal remedies, such as strychnin, is extremely doubtful.

It is very important to protect the anæsthetic part against external injuries. In anæsthesia of the buttocks and the heels, we must try in every possible way to prevent the formation of a bedsore. In anæsthesia of the trigeminus, we guard the eye as far as possible from the development of a neuro-paralytic keratitis by a carefully applied occlusive bandage, and treat even the slightest inflammation with the greatest care. We can thus almost certainly avoid bad results.

CHAPTER III

NEURALGIA IN GENERAL

Concept and Ætiology.—Although every pain is, of course, excited by abnormal irritation of the nerves, still, we are justified in giving a certain special variety of pain the name of neuralgia. By that name we understand pain due to a disease of a definite sensory nerve. The name neuralgia has, therefore, both a symptomatical and an anatomico-pathological significance. Symptomatically most neuralgias are characterized by the following peculiarities: 1, the pain is felt exactly in the course or in the distribution of one or more special nerve trunks or nerve branches; 2, it is usually of very considerable intensity; and, 3, as a rule, it is not present continuously, but it shows manifest variations. Many neuralgias come on in pronounced paroxysms of pain, which are interrupted by wholly painless intervals. Anatomically, neuralgia is not one single disease. Every severe affection of a sensory nerve leads to neuralgic pain. The trouble is often merely one symptom of another more extensive morbid process (symptomatic neuralgia). In other cases the affection of the sensory nerve is an independent disease by itself (idiopathic, primary neuralgia). In many idiopathic neuralgias we may with great probability assume an inflammatory affection of the nerve or the nerve sheath, as in sciatica, but in other cases, and indeed in most typical neuralgias, as in severe trigeminal neuralgia, this theory is improbable. In these cases the special nature of the "neuralgic disease" is still very obscure. At times, in separate attacks and without any known cause, extremely active irritative processes occur in the special nerve territory, causing the most intense pain. Trousseau has compared these paroxysms of sensory irritation to the motor discharges of the epileptic attack, and hence has spoken of "epileptiform neuralgia."

In regard to the general occurrence of neuralgia, age has thus far an influence that most cases occur in middle life; but trigeminal neuralgia occurs especially in older people. In children neuralgias are rarely seen. Sex exerts an influence thus far that certain forms, such as sciatica and brachial neuralgia, are more common in men. On the other hand, certain phases of the female sexual life, such as puberty, pregnancy, childbed, and the climacteric, favor the disposition to neuralgia. The general neuropathic predisposition, which is hereditary in the majority of cases, is of a certain significance. Neuralgia often appears in people who suffer from other neuroses, or in whose families other nervous diseases, such as the psychoses, epilepsy, hysteria, or neurasthenia, have repeatedly occurred. The physical constitution also seems to be of influence. It is frequently stated that we are most apt to see neuralgia in anæmic people, or in those whose constitution has been impaired by physical and mental strain, by imprudent living, or by mental excitement. We must, however, remark that many of the painful conditions considered in this class are not, strictly speaking, neuralgias.

We may mention, as exciting or at least as favoring causes of neuralgia: 1. Cold, the action of draughts, wind, or wet—the so-called "rheumatic neuralgias." It is not perfectly clear how the cold acts in such cases. We usually assume that by its action slight anatomical (inflammatory?) changes arise directly or in a reflex manner in the nerves themselves. 2. Mechanical and traumatic action. Among these are, first, wounds and contusions which directly involve the nerves. Thus, for example, extremely severe neuralgia sometimes arises from the pressure of foreign bodies, such as splinters of wood or of bones in wounds, on the branch of a nerve. We may also mention here the very severe neuralgia sometimes met with after amputations. These cases are probably due less to the so-called amputation neuromata than to the pulling and stretching of the nerve trunks in the amputation cicatrix, giving rise to the pain. In this class, too, belong many diseases in the neighborhood of nerves. Diseases of the bones or periosteum, by pressure and mechanical irritation, and often perhaps by the accompanying inflammation, may lead to neuralgia in those nerves which run through bony canals or grooves. Lastly, tumors, aneurisms, herniæ, or the gravid uterus, may lead to neuralgia by pressure on the neighboring nerves; but we must bear in mind that every pressure on a nerve does not lead to neuralgia in the same way, so that we must assume a special consequent change in the nerve in "neuralgia from compression." 3. The relation which certain infections and poisons have to the development of neuralgia is very important. In the first place, it is not impossible that many of the apparently "idiopathic" neuralgias are to be referred to infectious causes—an assumption which may be made, for instance, in intercostal neuralgia associated with an eruption of zoster (*q. v.*), or in many acute trigeminal neuralgias. Many neuralgias, however, also have a definite relation to other infectious diseases. We may mention here the malarial neuralgias, which are directly dependent upon the malarial infection, often come on at regular intervals, and are cured by specific treatment—namely, quinin. Neuralgia is often seen during the course, or as a result, of typhoid fever, smallpox, and similar acute infectious diseases, and in the secondary stage of syphilis. Among toxic substances we may mention especially alcohol, lead, arsenic, mercury, and perhaps nicotin, as those which have a relation to the development of neuralgia. Yet in these cases it is usually not true neuralgia, but pain of some other kind—the arthralgia of lead poisoning, the polyneuritic pains of alcoholic subjects, etc. 4. Symptomatic neuralgia is also found in many constitutional diseases, in gout, and very often in diabetes mellitus. It is supposed that in such cases abnormal chemical influences act on the sensory nerves. 5. Finally, there was formerly much said about "reflex neuralgia," where, for example, disease of the sexual organs was thought to cause neuralgia in distant nerves. Such observations, however, cannot endure strict criticism. Most of these cases are hysterical.

[Although the reflex origin of neuralgia and other nervous disturbances is very doubtful, it is well known that pain due to disease of a given organ may be referred to another part of the body. A familiar example is the reference to the knee of the pain from hip disease. This reference of pain to distant parts is probably dependent upon the anatomical distribution of the nerves. Head has made an exhaustive study of the distribution of the visceral nerves in the spinal cord, and has found that, in disease of the internal organs, the pain is apt to be referred to certain areas of the skin whose sensory nerves go to the corresponding spinal segments. The internal organs are represented in the spinal cord as follows: Heart, 1 to 3 D;[1] lungs, 1 to 5 D; stomach, 6 to 9 D (cardiac end, 6 to 7 D; pylorus, 9 D); intestines, 9 to 12 D; rectum, 2 to 4 S; liver and gall bladder, 7 to 10 D; kidney and ureter, 10 to 12 D; bladder (mucous membrane and neck), 1 to 4 S; overdistention of bladder, 11 D to 1 L; prostate, 10 to 12 D, 1 to 3 S, 5 L; epididymis, 11 D to 1 L; testicle and ovary, 10 D; appendages, 11 D to 1 L; uterus, in contraction, 10 D to 1 L; os uteri, 1 to 4 S. For the cutaneous areas corresponding to these segments of the cord, see the colored plate (Plate VI). Disease of the internal organs may also cause referred pains in the head, perhaps through the connections of the vagus nerve. Disease of the lungs causes referred pain in the fronto-nasal, mid-orbital, and fronto-temporal areas; of the heart in the mid-orbital, fronto-temporal, and temporal areas; of the stomach and intestines in the temporal, vertical, and parietal areas; of the liver in the vertical and occipital areas; and of the ovaries and testicles in the occipital area. Diseases of the uterus, Fallopian tubes, and bladder do not give rise to referred pain in the head. Affections in the head itself also give rise to referred pains in the head. Refraction errors cause pain in the mid-orbital region; disease of the cornea or anterior chamber of the eye, pain in the fronto-nasal area; of the iris, in the temporal area; farther back in the eye, in the temporal, fronto-temporal, and maxillary areas; of the membrana tympani and middle ear, in the hyoid area; elevation of tension in the middle ear. in the vertical and parietal areas; disease of the tongue, in the mental, hyoid, and superior laryngeal areas. Dana had previously represented the regions of referred pain in visceral disturbances in much the same way.—K.]

General Symptomatology of Neuralgia.—The neuralgic paroxysm begins either quite suddenly, or, more frequently, after certain prodromata, such as cold feelings, prickling, slight painful sensations, etc., have preceded it for some time. The pain during the attack is usually of the greatest severity, and is described as either burning and boring, or as shooting and tearing like lightning. There are frequently short temporary remissions of the pain. The location of the pain usually corresponds precisely to the distribution of the affected nerve, so that the patient can often point out quite definitely the anatomical course of the nerve. At the height of the attack there is often an "irradiation" or shooting of the pain into the territory of neighboring nerves. External irritants, such as cold air, mental excitement, and especially movements of the affected part, often produce an increase of the pain.

On physical examination we notice, first, certain disturbances of sensibility. The skin in the neuralgic part often shows more or less anæsthesia, which is most apparent in the intervals between the separate attacks and immediately after them. Much more frequently, however, both during the attack and during the time when the patient is free from pain, there is hyperæsthesia of the skin and the parts beneath. There are certain definite points which are often very sensitive and tender even to light pressure. These are called painful points (*points douloureux*). They were first fully described for the different forms of neuralgia by Valleix, in 1841, although in much too schematic a way, but they have some diagnostic importance, since they may often be found, not only during the attack itself, but also during the free intervals, although then they are less marked. The painful points always correspond to certain places in the course of the trunk or the larger branches of the affected nerve, and are found especially where, in marked and deep pressure on the nerve, we can press on some firm part beneath. They are probably always due to an abnormal sensitiveness to pressure in the affected nerve itself. In many cases of neuralgia they may be wholly wanting.

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[1] D, dorsal segment; L, lumbar; S, sacral.

Motor symptoms as well as sensory are not infrequent in neuralgia. Coexisting symptoms of paralysis must always be regarded as a complication caused by some coarse lesion of the motor nerves; hence, in pure idiopathic neuralgia they are entirely absent. The coexisting symptoms of motor irritation, which are often seen, are usually directly dependent upon the neuralgia, however, and hence are to be regarded as reflex contractions, due to the great irritation of the sensory nerves.

Vasomotor symptoms are often seen in neuralgia. In the face especially, in trigeminal neuralgia, we often see a marked pallor or a decided reddening of the skin and conjunctiva. Abnormal secretions of tears or sweat may also be seen during the attack or at the end of it. We do not know whether all these symptoms arise from direct or reflex nervous irritation. Trophic disturbances are noticed in various ways. During the attack we see eruptions of urticaria, or still more frequently of herpes vesicles, along the course of the affected nerves, as in herpes zoster. In severe and protracted neuralgias permanent changes in the tissues have been repeatedly seen in the parts supplied by the affected nerves. Among these are a falling out or a whitening of the hair, or more rarely an abnormally great growth of hair, thickening or atrophy of the skin, staining or pigmentation of the skin, atrophy of the deeper parts, etc. Lastly, we may mention that during the neuralgic attack we sometimes find a diminished frequency of the pulse.

The general nutrition of the body in neuralgia is especially impaired when sleeping and eating are constantly disturbed by the attacks of pain. Then the disease gradually has a noticeable action on the whole constitution. The patient becomes pale and emaciated, and often the persistent and distressing pain is not without influence on his mental condition. He becomes irritable and inclined to melancholy. Patients have, in repeated instances, even committed suicide as a result of severe and incurable neuralgia.

The whole course of neuralgia shows the greatest diversity. As has been said repeatedly, the onset of the disease in separate attacks is the chief characteristic feature. The precise pathogenesis of these attacks is, of course, entirely unknown to us. These attacks usually come on every day or many times a day, or sometimes at longer regular or irregular intervals. They may last only a few minutes or several hours. During the period between the attacks many patients feel quite well, but some have still a certain sensitiveness of the skin. The disease as a whole sometimes lasts only a few days or weeks, but sometimes it persists with manifold variations for years and years, and, in a word, is not capable of improvement; although, on the other hand, there are recoveries after the disease has lasted for years. In many cases, of course, the disease depends upon the presence of some definite anatomical cause, such as a tumor, a disease of the bone, or an aneurism.

Many details will be spoken of in the following chapter.

General Treatment of Neuralgia.—In treating neuralgia itself we must always look first with great care for some causal factor, which may be accessible to treatment. This fulfillment of the causal indication is possible, especially in neuralgias which are due to mechanical causes. The extirpation of tumors, the excision of cicatrices, the removal of foreign bodies, the treatment of inflammatory new growths, of syphilitic affections, of aneurisms, etc., is occasionally attended by the most brilliant success, but, of course, in many other cases the underlying disease is unfortunately not amenable to successful treatment. We should also carry out a causal treatment in the neuralgias which are to be referred to general anæmia, to a general neuropathic constitution, to hysteria, etc. In such cases we must always ascribe great value to the general treatment, such as diet, manner of life, psychical treatment, baths, iron, and nervines, as well as to the special treatment directed against the neuralgia; and the same holds true, of course, in the neuralgias occurring in diabetes, gout, and syphilis. We must always bear particularly in mind the possibility of syphilitic neuralgia, since in such cases iodid of potassium may often have a marked curative action. We may also fulfill the causal indication in the malarial neuralgias. If neuralgia comes on at approximately regular intervals in persons who come from a malarial district, and who perhaps have already suffered from other malarial affections, the exhibition of quinin in large doses, 20 to 45 gr. (gm. 1.5 to 3) at once, will often cut short the attack. In obstinate cases in which quinin does no good, we should try arsenic, in the form of Fowler's solution. In many apparently toxic neuralgias, too, from lead, mercury, or alcohol, our first endeavor in treatment should be to remove the cause of the disease.

In all cases where the causal treatment cannot be carried out, or where it alone is not sufficient, we must consider those numerous remedies and methods of treatment which correspond to the *indicatio morbi* and to the symptomatic indications. Starting with the hypothesis of an inflammatory condition of the nerve, the attempt is often made to exert a favorable influence on the disease by local derivatives, mustard plasters, irritating embrocations, such as spirit of mustard, veratrin ointment (two and a half per cent), or tincture of iodin, or by blisters, or even by the cautery. The remedies first mentioned are used only in mild cases. Vesicatories, placed along the course of the affected nerve, or behind the ear in facial neuralgia, sometimes act very well in fresh cases, especially in fresh "rheumatic" cases of sciatica. We resort to the hot iron chiefly in old and very severe cases, in which, indeed, especially in sciatica, some very favorable results have been claimed.

The local electrical treatment of neuralgia is sometimes of undoubted benefit. We many times secure an improvement in the symptoms, which is, of course, temporary, even in those cases where the special cause of the disease is which is most apparent in the intervals between the separate attacks and immediately after them. Much more frequently, however, both during the attack and during the time when the patient is free from pain, there is hyperæsthesia of the skin and the parts beneath. There are certain definite points which are often very sensitive and tender even to light pressure. These are called painful points (*points douloureux*). They were first fully described for the different forms of neuralgia by Valleix, in 1841, although in much too schematic a way, but they have some diagnostic importance, since they may often be found, not only during the attack itself, but also during the free intervals, although then they are less marked. The painful points always correspond to certain places in the course of the trunk or the larger branches of the affected nerve, and are found especially where, in marked and deep pressure on the nerve, we can press on some firm part beneath. They are probably always due to an abnormal sensitiveness to pressure in the affected nerve itself. In many cases of neuralgia they may be wholly wanting.

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The local electrical treatment of neuralgia is sometimes of undoubted benefit. We many times secure an improvement in the symptoms, which is, of course, temporary, even in those cases where the special cause of the disease is not influenced by the electricity, although in idiopathic neuralgias in fresh, and sometimes even in old cases, we can often obtain a complete cure. There are no general rules with regard to the methods to be employed, since different specialists have their own favorite methods. The following forms of application are most in use and are most to be recommended: 1. Stabile action of the anode of a constant current on the affected nerve trunk over as great an extent as possible, especially on any painful points. We must entirely avoid any great variations in the current or interruptions of it. We gradually increase the intensity of the current up to medium strength. The sittings should last three to six minutes, and sometimes even longer, and they must be repeated daily. 2. In neuralgia of the larger nerves we should use a stabile descending (sometimes ascending) constant current, in which the anode is placed on the most central point of the nerve trunk available, or on the vertebral column, and the cathode on different peripheral points. 3. The faradic current also frequently acts very well. We faradize the nerve either with a moderately strong "increasing" current, or we apply the wire brush to the skin over the affected nerves. The latter method is very painful, but is often attended with excellent results. As a rule, it is always well to begin with a mild and very cautious use of electricity, and not to go on to the stronger currents until later. Electricity often acts brilliantly at once, during the attack of pain, but sometimes no improvement is seen until after several sittings. If, after two or three weeks, we obtain no result at all, after employing different methods of applying electricity, it is the best plan to give up electrical treatment entirely as not suited to the case. The treatment of true neuralgias with static electricity, D'Arsonval currents, electric light, electro-magnetic currents, etc., is scarcely ever attended with success. The numerous reported cures refer in most cases probably to hysterical pains.

In the treatment of neuralgia we must consider a number of internal remedies, as well as electricity; some of these act symptomatically, like the narcotics, while others have obtained the reputation of having a specific action. Among the latter, quinin has decidedly the greatest value. In severe cases quinin may do excellent service, not only in malarial neuralgias, although most surely in those, but also in "idiopathic" neuralgias. It is in these cases that the remedy is given in fairly large doses. We begin with 15 to 30 gr. (gm. 1 to 2) a day, best given in one dose, and in severe cases we may increase to 60 or 75 gr. (gm. 4 to 5) or more. We see the best results from quinin in trigeminal neuralgia, while in other forms, as in sciatica, it is of little value. Besides quinin, salicylate of sodium is sometimes beneficial, especially in the recent rheumatic forms of neuralgia. Antipyrin, in 15- to 30-gr. (gm. 1 to 2) doses; aspirin, in 2-gr. (gm. 0.1) doses [or more] several times daily; pyramidon, in 4- to 8-gr. (gm. 0.25 to 0.5) doses; phenacetin, in 7- to 15-gr. (gm. 0.5 to 1) doses; salipyrin, in 20- to 30-gr. (gm. 1.5 to 2) doses; and similar remedies, have lately been very often used. Next in rank, for continued use, come arsenic and bromid of potassium. The former is given in pill form or as Fowler's solution, five drops three times a day, increasing gradually. Bromid of potassium acts only in large doses, 45 to 75 or 150 gr. (gm. 3 to 5 to 10) a day.

In all severe neuralgias the use of narcotics, especially of morphin, is unavoidable. Morphin is used almost exclusively during the attack, and is best given as a subcutaneous injection of $\frac{1}{12}$ to $\frac{1}{6}$ gr. (gm. 0.005 to 0.01) in the vicinity of the painful part. The anodyne effect almost invariably follows, but in obstinate and protracted cases the patient gradually becomes accustomed to the remedy. We must then resort to still larger doses, and these, too, finally fail in their effect. Among the victims of the morphin habit we find many patients who have suffered or who still suffer from severe neuralgia, so that we must always be very cautious and conservative in the protracted use of morphin. We should be especially cautious before deciding to put the hypodermic syringe into the patient's own hand. Many physicians ascribe not only a palliative but a permanent benefit to injections of morphin in neuralgia. We sometimes see, in fact, that mild neuralgias recover under the exclusive use of morphin injections; but these are probably cases of spontaneous recovery. The internal use of morphin and opium preparations is decidedly inferior to the subcutaneous administration in certainty and rapidity of action.

Various hypnotics (chloral hydrate, veronal, sulphonal, etc.) are frequently employed in severe neuralgias, but their effect is decidedly inferior to that of morphin. The external application of narcotic ointments, or embrocations, is much employed in practice, but it appears to be of advantage only in milder cases. We prescribe ointments with extract of opium (1 to 10), extract of belladonna (2 to 10), extract of opium and veratrin (1 part of each to 20 of simple ointment), etc. We may also use chloroform, by applying blotting paper wet with chloroform, or embrocations of chloroform oil, and ether as a spray.

Recently the so-called injection treatment with [alcohol] stovain and similar drugs has attained great practical significance. It will be considered in detail in the discussion of the different forms of neuralgia.

In severe cases the surgical treatment of neuralgia is often of great importance—the section of nerves, neurotomy, or the excision of a portion of the nerve, neurectomy—in order to prevent the union of the divided nerve, or, finally, the drawing out of the nerve—so-called exeresis. A detailed account of the methods of operation is to be found in the text-books of surgery. We may state here merely that the surgical treatment of neuralgia, especially of severe trigeminal neuralgia, often has excellent results. The explanation of this success, however, is by no means easy. There is probably not only a break in conduction from the pain-exciting spot to the center, but a secondary change in the whole sensory neuron, by which the neuralgic change is made to cease. Of course a cure is not obtained in all cases. In any event, we should advise an operation only in severe cases, where all other remedies have been tried in vain, and the patient may be promised the possibility, or even the probability, of success, but never the certainty of it. Besides section of the nerves, nerve stretching has been frequently tried in neuralgia. We will return to this method of treatment in speaking of sciatica.

The chief question as to the use of hot air, baths, etc., arises only in the treatment of neuralgias in the domain of the nerves of the extremities, especially in sciatica, and therefore these and massage will be spoken of more in detail in connection with the separate forms of neuralgia.

CHAPTER IV

THE INDIVIDUAL FORMS OF NEURALGIA

1. NEURALGIA OF THE TRIGEMINUS

(Prosopalgia. Tic douloureux. Fothergill's Face-ache)

Ætiology.—Trigeminal neuralgia is one of the commonest and most important neuralgias. In its origin many causes and predisposing factors of all sorts play a part, as was stated in detail in the preceding chapter. Many mild forms, especially neuralgia in the first branch, apparently are of acute origin and have no discoverable cause. In such cases some unknown infectious or autotoxic influences may play a part. Not infrequently, especially in mild neuralgias of the first branch, we cannot overlook the influence of exposure to cold (wind, wet) as a possible cause. Neuralgias after acute infectious diseases (influenza, typhoid), and especially malarial neuralgias, are situated most frequently in the first branch of the trigeminus, more rarely in the second, and only exceptionally in the third. Trigeminal neuralgia may also be due to diseases of the cranial bones and periosteum (tumors), very often to diseases of the teeth (caries, exostoses, anomalies of development and position of the teeth), and also to diseases of the nasal and frontal cavities and diseases of the middle ear and of the eye (inflammation and also strain of the eyes). Romberg found an aneurism of the internal carotid, pressing on the Gasserian ganglion, as the cause in a severe and incurable case. We have seen a precisely analogous case. Finally, the severe trigeminal neuralgias, which last for years, are of very great practical importance; for these we can usually find no cause at all, or at most a general nervous predisposition. This form, which is the prototype of the most frequent genuine, severe, idiopathic neuralgias, comes on usually in somewhat advanced life, and is about equally frequent in women and men. The special form of disease of the nerve in these cases is still wholly obscure. The ætiology of this painful disease is almost entirely unknown, since the anatomical findings from the resected pieces of nerves and extirpated Gasserian ganglia do not permit of any definite interpretation. As arteriosclerosis is frequently present, we are very much inclined to assume some connection with this lesion in many of the cases. The arteriosclerotic changes in the small blood vessels which supply the Gasserian ganglion or the nerve branches, can very probably cause a disturbance of nutrition in the nerve tissue, and in this way produce these intense pains. [Dana, Putnam, and others have found degenerative neuritis in the peripheral fibers of the excised nerve in these severe cases, sometimes associated with obliterating endarteritis in the blood vessels supplying the nerves. Spiller has found extensive degenerative changes in the nerve cells of the Gasserian ganglion, but these may be secondary to the changes in the nerve fibers.—K.]

Symptoms and Course.—The attacks of pain in neuralgia of the fifth pair are usually quite intense, and in severe cases they may attain a most distressing and terrible severity. They arise either entirely without cause, or from some slight influence, such as washing the face [making the slightest movement of the face in eating or talking.—K.], taking physical exercise, or mental excitement. They often come on quite regularly at definite hours, especially in the forenoon, but in other cases they follow no rule. There are sometimes long, painless intervals between the separate attacks, and sometimes the attacks follow one another in an almost uninterrupted series. The lightning-like, cutting pains extend to the distribution of the affected branch of the trigeminus, but they sometimes shoot into the occiput, the back of the neck, the shoulders, etc. We can often perceive reflex twitchings in the face, especially blepharospasm, and twitching of the corners of the mouth. The vasomotor disturbances are noticed at first as abnormal pallor, but later usually as quite an abnormal redness of the face and conjunctiva. In neuralgia of the upper two branches we often see, during the attack, an unusually great secretion of tears. An excessive flow of saliva and an increased secretion from the nasal mucous membrane are more rare. Sometimes, but still quite rarely, we see herpetic eruptions in the course of the affected nerve, zoster frontalis, herpes of the conjunctiva, etc. In some cases, too, more severe diseases of the eyes, belonging to the category of neuroparalytic ophthalmia (*vide* page 161), have been observed, and also paralysis of the ocular muscles. Such complications probably always point to some gross organic lesion, generally a tumor of the base of the brain. In neuralgias that have lasted longer, we often see still further trophic disturbances: changes (thickening or atrophy) in the skin and subcutaneous cellular tissue, the hair turning gray or falling out in the frontal region, etc.

Most trigeminal neuralgias are situated, not in the whole distribution of the nerve, but only in one or more of its branches (see Figs. 122 and 123, page 161). We accordingly distinguish: (1) *Neuralgia of the first branch* (*ophthalmic neuralgia*), which is especially frequent as supraorbital or frontal neuralgia. The pain shoots chiefly from the supraorbital foramen upward into the forehead, and sometimes to the region of the internal angle of the eye and the bridge of the nose (ethmoidal nerve), or into the zygomatic region (lachrymal nerve). In this we find, as a rule, that pressure on the point of exit of the nerve at the supraorbital foramen is more or less painful. More rarely we find painful points also on the nose, at the inner angle of the eye, or the parietal eminence, etc. (2) *Neuralgia of the second branch* (*supramaxillary neuralgia*) is most frequent in the distribution of the infraorbital nerve (infraorbital neuralgia), with the chief painful point at the infraorbital foramen, and others on the zygoma, on the upper lid, etc. The chief pain usually starts from the infraorbital foramen, and is situated in the lower eyelid, the cheek, the upper jaw, the upper lip, and in part also in the temporal region. During the attack of pain tears are often seen in the eye. (3) *Neuralgia of the third branch* (*inframaxillary neuralgia*) is situated chiefly in the branches of the mandibular nerve (inferior alveolar and mental nerves). The pain affects the region of the lower jaw and the chin; but sometimes the pain occurs in the temporal region, in the auriculo-temporal nerve, and in some cases it is especially distressing in the tongue, in the lingual nerve. The chief painful points are at the mental foramen and the lower temporal region.

The general course of neuralgia of the fifth pair differs very much in different cases. We see all forms, from the mildest, which rapidly passes off, to the severest and incurable types, which may drive the patient to despair. The milder forms, such as occur with colds, acute infectious diseases and similar causes, are generally located in the upper division (n. frontalis). Under proper treatment they usually get well in a few weeks, but often there is an evident tendency to relapse. The severe forms, which continue for years (the tic douloureux proper), occur rarely in the first division, more frequently in the second and third divisions. Occasionally two, or even all the branches, are affected simultaneously. Neuralgia of the third division, involving the tongue, is very correctly considered to be particularly severe.

Diagnosis.—The diagnosis of trigeminal neuralgia is, in general, not difficult if only we consider the character and distribution of the pain, its occurrence in separate paroxysms, and the presence of any painful pressure points. Of course a careful local examination is always necessary to exclude the existence of inflammatory or other diseases of the bones, periosteum, and teeth. Neuralgia of the first branch may be confounded with other forms of headache, and especially with true migraine (*vide infra*), since in the latter the pain is often located in the region of the supraorbital nerve. Only by a careful consideration of accompanying symptoms (scotoma, vomiting) can we avoid a mistake. In the severe neuralgias of the second and third branches the reflex muscular twitchings are very marked, so that, on very superficial observation, trigeminal neuralgia may be mistaken for clonic facial spasm ("tic douloureux" for "tic convulsif").

Prognosis.—The prognosis is never to be made with absolute certainty. It is most favorable in fresh cases of supraorbital neuralgia which have, as a basis, a manifest cause, which may be removed; but if the affection is due to a coarse anatomical cause which cannot be removed, or if we are dealing with chronic cases, especially of the second or third branch, the prognosis, unfortunately, is often very doubtful or utterly unfavorable.

Treatment.—The treatment of trigeminal neuralgia rests entirely upon the principles given in the preceding chapter. In searching for the causal indications we must look chiefly, in neuralgia of the second and third branches, for diseased teeth, and also in all cases for any affections of the nose, of the frontal sinuses, or of the middle ear. If such affections exist they should be treated in the appropriate way. Carious teeth, which are painful and which seem to have a relation to the neuralgia, should always be removed; but we must warn patients against permitting in their despair all their sound teeth to be extracted by unskillful dentists without any benefit—a thing which we have frequently known to happen. If there be a suspicion of syphilis or malaria, we should prescribe iodid of potassium or quinin. [Even in non-syphilitic cases of the severer type large doses of iodid of potassium (gm. 2 to 6 (gr. 30 to 90, thrice daily) combined with mercury may act very beneficially. Aconitin and aspirin are of some benefit. The application of the positive pole of the galvanic battery, wet with a ten- to twenty-per-cent solution of cocain or chloroform, may give temporary relief (electrical cataphoresis). Dana has obtained good results from absolute rest in bed and daily injections of strychnin (gm. 0.001 to 0.004, gr. $\frac{1}{60}$ to $\frac{1}{15}$) for several weeks.—K.]

In addition, in fresh "rheumatic" neuralgias, we should first try quinin, in doses of 15 to 30 gr. (gm. 1 to 2) or more, or one of the many new "nervines"—antipyrin, aspirin, pyramidon, phenacetin, salipyrin, exalgin, trigemin, etc. They sometimes act very favorably, but in other cases the action is uncertain. Mixtures of the above-mentioned drugs (antipyrin with phenacetin, etc.) often act better than a single drug. Many other remedies, which were formerly often used in neuralgia, are at present only rarely prescribed, as, for example, butyl (croton) chloral, tincture of gelsemium, aconitin, etc. Arsenic is more frequently prescribed at present, with somewhat more prospect of benefit.

Besides internal remedies, electricity is also to be considered. It is best given by applying the anode to the painful points of exit of the affected nerve and to any other painful points, while the cathode is held on the back of the neck. We should use weak currents and avoid any sudden variations in the current. Other methods (faradic brush) may also be tried. Massage has also been recommended for trigeminal neuralgia, but in really severe cases neither massage nor electricity is of any permanent benefit.

If a severe neuralgia persists in spite of rational treatment by electricity or drugs, I recommend, in the first place, to try the injection treatment. In several cases I have seen remarkably good results, which were obtained by the injection of 3 to 15 minims (gm. 0.2 to 1) of a one-per-cent solution of osmic acid at the point of exit of the painful nerve. Lately, we have mostly employed a eucain-sodium-chlorid solution, injecting from ʒiv to ʒv (15 to 20 c.c.) at the points of exit of the painful nerves. Personally I have had some very favorable results with this method in severe cases of trigeminal neuralgia. A more detailed exposition of this treatment will be found under the treatment of sciatica. With Schlösser's method of alcohol injection for the treatment of neuralgia, we have had no personal experience [but in many cases it has caused a cessation of the pain for a considerable period.—K.] If the injection treatment prove unsuccessful, we should not delay too long in proposing to the patient operative treatment. In frontal and infraorbital neuralgia especially neurectomy is a comparatively slight operation, which has many excellent results to show. Even in the severe and otherwise incurable neuralgias of the third branch neurectomy is sometimes attended with the best results. The full description of the different operations must be left to the text-books on surgery. We may add that Krause and others in very severe cases have extirpated the Gasserian ganglion successfully and with benefit.

If we have to limit ourselves to a purely symptomatic treatment, injections of morphin become unavoidable in continued severe cases, but we should restrict their use as much as possible. The above-mentioned nervines have sometimes a symptomatic action. We can sometimes lessen or shorten the paroxysm of pain by pressure on the nerve or by compression of the carotid.

2. OCCIPITAL NEURALGIA

Of the neuralgias involving the sensory region of the upper four cervical nerves, neuralgia of the occipitalis major (Figs. 122, 123) is relatively the most frequent and practically the most important; but true primary occipital neuralgia (from catching cold, acute infectious diseases, mechanical injuries from carrying weights on the head and neck, etc.) is extremely rare. Secondary neuralgias from affections of the upper cervical vertebræ (caries, arthritis deformans, neoplasms) are somewhat more often met with. The not infrequent hysterical occipital pains are generally readily distinguished from true neuralgias on careful examination.

Occipital neuralgia is usually located in both occipital nerves at once, although often more severe on one side than on the other. Painful points are most frequently found midway between the mastoid process and the upper cervical vertebræ. Vasomotor disturbances, falling out of the hair, etc., have been often observed.

The prognosis depends upon the variety of the disease. In primary neuralgic affections it is generally favorable. In such cases, if fresh, a rapid recovery may often be brought about by aspirin, antipyrin, the constant current, or sometimes by strong cutaneous irritants to the back of the neck (blisters). In other cases, of course, which are due to some severe disease (vertebral caries, tumors, etc.), the prognosis is often utterly unfavorable, and treatment can be only symptomatic (morphin, etc.).

All the other neuralgias in the distribution of the cervical plexus are rare. They occur in the distribution of the occipitalis minor (when, according to Seeligmüller, they are quite frequently due to syphilis, and are cured by iodid of potassium), and in the distribution of the great auricular, subcutaneus colli and the supraclavicular nerves. A phrenic neuralgia even has been described, in which the pain extends along the course of the phrenic nerve to the insertion of the diaphragm, but this is at all events very rare. In these cases, as in many of the so-called visceral neuralgias, we probably mistake quite different forms of disease (especially diseases of the heart and blood vessels) or hysterical symptoms for neuralgia.

3. NEURALGIA IN THE REGION OF THE BRACHIAL PLEXUS

(Cervicobrachial Neuralgia)

Brachial neuralgia is, on the whole, rare, and it is hardly ever limited strictly to the distribution of a single nerve. In general the radial and ulnar nerves are rather more frequently affected than the median. Neuralgia of the internal cutaneous nerve and other cutaneous branches, and neuralgia of the sensory branches of the axillary [circumflex] nerve, have also been described. In regard to ætiology, we have to mention, first, wounds and contusions of the nerves, and also cicatrices and foreign bodies. The amputation neuralgias also belong to this class. Severe neuralgia in which the pain may extend over a greater part of the whole arm is sometimes seen after injury of the fingers (crushing, cuts, etc.). In some of these cases we may have to do with an ascending neuritis, starting from injury of a small branch of a nerve; in other cases there are probably cicatricial contractions, thickenings of the neurilemma, or small neuromata developing after the injury, which excite the pains. With all these traumatic and reflex neuralgias, we must always bear in mind the possibility of a hysterical—i. e., purely psychical—pain; it is certain that many cases belonging to this category were formerly incorrectly interpreted. The "amputation neuralgias," which are often very severe in amputation stumps, are due to neuromata which form on the cut ends of nerves. Severe neuralgia often arises from pressure on the brachial nerves, as in cases of tumors in the axilla or neck (cancer, etc.), aneurisms of the aorta, etc. Double brachial neuralgia should always excite suspicion of disease in the neighborhood of the upper posterior nerve roots, especially of cervical pachymeningitis, spondylitis of the lower cervical vertebræ, meningeal tumors, etc.

If we except the neuralgias secondary to trauma and compression already described, true primary neuralgia in the region of the brachial nerves is very rare. We may sometimes, and most frequently, speak of rheumatic neuralgia coming on after exposure to cold, although the distinction between this form of neuralgia and true neuritis (*q. v.*) is often very slight. If we hold less strictly to the conception of neuralgia, and include also the "nervous" pains not limited to the region of one special nerve and not due to any other discoverable local disease, we may find such "brachialgias" more frequently. They come on chiefly as one symptom of hysteria or neurasthenia in persons with a nervous predisposition, and are often very obstinate and apparently severe. We must also pay especial attention to the patient's calling and occupation. In seamstresses, pressers, piano players, persons who write much or do much other work with the arms supported, enthusiastic tennis players, etc., we sometimes see quite severe brachialgia for which we can find no other cause —the so-called "occupation neuralgias." A general nervous predisposition also comes into play not infrequently in such cases.

We must mention finally those brachialgias which are dependent upon arteriosclerosis. Recently several observations have been made which indicate that entirely analogous disturbances in consequence of arteriosclerotic changes can occur in the arms, as have been described for the legs under the name of "intermittent claudication," or dysbasia arteriosclerotica (*vide* page 458). In the true stenocardiac attack (*vide* page 418) the pain often shoots into the left arm. Many brachialgias, apparently associated with gout or diabetes mellitus, may be dependent upon such vascular changes and the disturbances of nutrition caused by them in the peripheral nerves. It is possible that the diseased vessels themselves may give rise to painful sensations.

Symptoms and Diagnosis.—We have little to add concerning the special symptomatology of brachial neuralgia. The pain, as we have stated, is usually ascribed to the whole course of the nerves without being very exactly localized. It is greater either in the upper arm or in the forearm and fingers. Painful points are sometimes found over the brachial plexus, over the radial nerve on the external surface of the upper arm, over the ulnar nerve in the sulcus at the internal condyle, over the median nerve at the inner border of the biceps, and where the cutaneous nerves emerge from the fasciæ. Vasomotor and trophic disturbances, such as "glossy fingers," a peculiar, shiny, atrophic condition of the skin of the fingers, eruptions of herpes, etc., are often seen in true organic neuralgias, and sometimes we also see a pronounced atrophy of the whole arm. The diagnosis of neuralgia as such is usually easy; we must bear in mind only the risk of confusion with articular affections, and especially with beginning arthritis deformans. It is often much harder to determine the cause of the neuralgia. This always requires a careful general investigation and close attention to all the above-mentioned conditions which may be involved.

Treatment.—The treatment of brachial neuralgia is often no easy task, since we frequently have to do with protracted and obstinate diseases. The causal indications should be met if possible; besides these we should make use chiefly of electricity (the descending galvanic current along the affected nerves). To relieve the pain we may also use salicylate of sodium, antipyrin, phenacetin, etc., and finally local warm packs, peat baths, sand baths, etc. We should be very guarded in using narcotics (morphin) except in severe hopeless cases. If the brachialgia be associated with neurasthenia or hysteria, we can obtain success only by an appropriate general treatment (hydrotherapy, Scotch douches, electrical applications to the entire body, etc.) with a psychical suggestive influence. In some severe cases of true neuralgia we must consider the possibility of surgical interference, such as nerve stretching, the extirpation of cicatrices, etc.

4. INTERCOSTAL NEURALGIA

(Dorso-intercostal Neuralgia)

The neuralgias of this class are almost always pure intercostal neuralgias, since the posterior dorsal branches of the thoracic nerves are only exceptionally involved. The middle intercostal nerves, from the fifth to the ninth, are usually affected, one or more of them being attacked at the same time. The affection is much more frequent on the left side than on the right.

Ætiology.—In regard to ætiology, it is important to remember that obstinate intercostal neuralgias are often a symptom, and for a long time the only symptom, of severe organic disease, especially affections of the ribs; diseases of the vertebræ, such as caries, carcinoma, and actinomycosis; diseases of the cord, such as tabes, meningeal tumors, syphilis; and aneurism of the aorta. Genuine idiopathic intercostal neuralgia, however, is also met with, as well as these symptomatic forms, but, according to our own experience (with the exception of herpes zoster), much less often than we might imagine from many of the statements. Intercostal neuralgia has been reported, especially in anæmic and nervous women, both in youth and middle age. Traumatic lesions of the intercostal nerves and taking cold also play a part in the ætiology.

The pain in intercostal neuralgia may attain a remarkable severity, and it is usually increased by any considerable movement of the thorax. Hence the patient avoids deep inspirations, coughing, loud talking, etc., as much as possible. We usually find three painful points—one near the vertebral column, one somewhere in the middle of the nerve, and a third near the sternum or over the rectus abdominis. We may mention, among the trophic disturbances, the comparatively frequent occurrence of herpes zoster. The pain precedes the eruption of zoster, or comes on at the same time with it. It often lasts for a long time after the cutaneous affection has healed. The special method of origin of the zoster vesicles is still obscure. A number of careful anatomical investigations (A. Dubler) favor the hypothesis that there is a genuine intercostal neuritis as the primary cause of the herpes zoster, and that the formation of vesicles arises simply from a direct extension of the inflammatory process from the terminal branches of the nerves to the skin. The hypothesis of special trophic disturbances would therefore be unnecessary. On the other hand, in numerous cases of herpes zoster, Head and Campbell have recently demonstrated an acute, generally hemorrhagic inflammation of the corresponding spinal ganglia. It is worthy of note that the attacks of zoster often exhibit a certain epidemic, and sometimes even an endemic distribution, so as to suggest an infectious agency. The almost constant swelling of the neighboring lymph-glands in the axillæ, at the lower border of the pectoral muscle, etc., also favors the theory of the existence of a true inflammatory process in herpes zoster. We can often find a slight diminution of sensibility on the skin between the individual vesicles.

Course of the Disease.—The course of intercostal neuralgia depends chiefly upon the ætiology of the affection. Primary neuralgias are often quite obstinate, but on the whole they usually give a favorable prognosis. This is especially true of the intercostal neuritis associated with herpes zoster. The differential diagnosis between genuine intercostal neuralgia and rheumatic affections of the muscles, incipient pleurisy, etc., is not always easy. In these cases a careful physical examination, a consideration of the localization of the pain, of the presence of painful points, and of the whole course of the disease, are needed to protect us from errors. Stenocardiac attacks (see page 418) are sometimes falsely regarded as intercostal neuralgia, and we must also remember that severe diseases of the vertebræ or the spinal meninges may at first assume the aspect of intercostal neuralgia.

Treatment.—The treatment is governed by the general rules given in the previous chapter. Cutaneous irritants, mustard plasters, or blisters often act very well in fresh cases. Local anæsthesia (chloroform, ether), especially a spray of methyl or ethyl chlorid, may occasionally be of benefit. Electricity is given by the faradic brush or the constant current; with the latter, the cathode is placed on the vertebral column, and the anode on the lateral and anterior painful points, using quite a strong stabile current. All the usual antineuralgics (antipyrin, phenacetin, exalgin, etc.) may be used internally. In severe cases we cannot avoid injections of morphin. Injections of eucain should, by all means, be tried. Herpes zoster heals by simple treatment with lanolin or by dusting on powders, such as 1 part of zinc oxid to 2 of starch.

Mastodynia (*Neuralgia of the Mammary Gland*).—Mastodynia ("irritable breast" of Astley Cooper) is to be considered as a special form of nerve pain in the distribution of the intercostal nerves. It occurs almost solely in women, after the age of puberty, although it may occur in men, and is a very painful, distressing, and obstinate affection. The pain is either continuous, or it comes on in separate paroxysms, sometimes accompanied by vomiting. The whole breast, or more frequently different parts of it, is extremely sensitive to the touch. We know little that is definite as to its ætiology.

The cases which I have seen myself all belonged undoubtedly to the domain of hysteria, and could generally be traced to a psychical disturbance—namely, the fear of cancer of the breast. This dread occurs particularly where there are small painful nodules (so-called tubercula dolorosa, neuroma?) to be felt in the mammary gland.

The affection may last for years. Favorable results can be secured only when psychical causes are present which can be removed by appropriate suggestions. If such causes do not exist, warm packs to the breast, bandaging the breast, and especially narcotics, may afford relief. Electricity may occasionally be of decided service. In the worst cases operative interference has been attempted—amputation of the breast, or extirpation of the painful nodules—but its results are uncertain. Nägeli highly recommends stretching of the whole breast *en masse;* the breast is seized with both hands and stretched and extended for several minutes with special regard to the tenderest points.

5. NEURALGIA IN THE REGION OF THE LUMBAR PLEXUS

As the neuralgias of this class are rare and show few peculiarities, we will content ourselves with a brief account of the most important forms.

Lumbo-abdominal neuralgia causes pain in the lumbar region, which shoots into the buttocks, the hypogastrium, and the genitals. Crural neuralgia is seated, in part, in the region of the external anterior cutaneous nerve of the thigh, and in part in the region of the cutaneous branches of the crural nerve, the internal and middle cutaneous. Its distribution over the cutaneous distribution of the great saphenous nerve, the inner portion of the calves, and the inner border of the foot, is especially characteristic. In obturator neuralgia the pain extends along the inside of the thigh, down to the vicinity of the knee joint (see Figs. 120 and 121, page 160).

In their individual characteristics, all these neuralgias agree with what has been said in the previous chapter. The diagnosis is not always easy, and we must pay special attention to avoid confusing them with affections of the bones and joints, with lumbago, renal colic, etc.

We must mention here a special symptom-complex, *meralgia parœsthetica* (Bernhardt, Roth), the chief symptoms of which are pain, paræsthesia, and partial anæsthesia on the outer side of one or both thighs—that is, in the territory of the external cutaneous femoral nerve. Mechanical factors in particular (long-continued walking, the pressure of articles of clothing, hard work), or in some cases exposure to cold or acute infectious diseases, seem to excite the trouble, which is sometimes very persistent. The treatment consists of embrocations, electricity, baths, etc.

6. SCIATICA

(Ischiatic Neuralgia. Ischialgia. Malum Cotunnii)

Ætiology.—Next to trigeminal neuralgia, sciatica is by far the most frequent and practically the most important form of neuralgia. The anatomical course of the sciatic nerve renders it especially exposed to mechanical injuries and the influence of cold. Many cases of sciatica are certainly due to such causes as direct trauma, overexertion of the leg in hard work, sitting for a long time on an uncomfortable seat, etc.; and this is why sciatica is in general far commoner in men than in women, and also much commoner in the laboring classes than in the higher classes. Certain ætiological conditions may, of course, provoke sciatica in women also, especially the pressure of the gravid uterus on the sacral plexus, or mechanical injury to the plexus in difficult labor (forceps, etc.). Exposure to wet and cold appear to play a fairly important rôle in the ætiology of sciatica. Venous stasis in the pelvic veins (hemorrhoids) and habitual constipation more rarely seem to give rise to the development of sciatica.

The relations of sciatica to certain constitutional and toxic general diseases are of practical importance. Thus, a simple or even a double sciatica is not very rare in diabetes mellitus, and it is sometimes seen in true gout (arthritis uritica), in articular rheumatism, following syphilis or gonorrhea, in alcoholic subjects, etc. We must also bear in mind the occurrence of apparently pure sciatic pain in tabes dorsalis.

Symptomatic neuralgia in the region of the sciatic nerve is also seen in pelvic tumors, caries of the sacrum, and similar diseases.

Symptoms and Course.—Sciatica takes a somewhat different position from the other forms of neuralgia, since the sciatic pain does not usually show the special characteristics of true neuralgia in a fully pronounced fashion. In particular, the individual paroxysms of pain are rarely so pronounced or so interrupted by free intervals in sciatica as is the case, for example, in many trigeminal neuralgias. Although an increase or a diminution of the pain is common in sciatica, the pain, on the whole, gives us more definitely the impression of a persistent neuritic pain, and we can therefore scarcely be mistaken if we consider the anatomical cause of most cases of ordinary sciatica to be an actual sciatic neuritis or perineuritis.

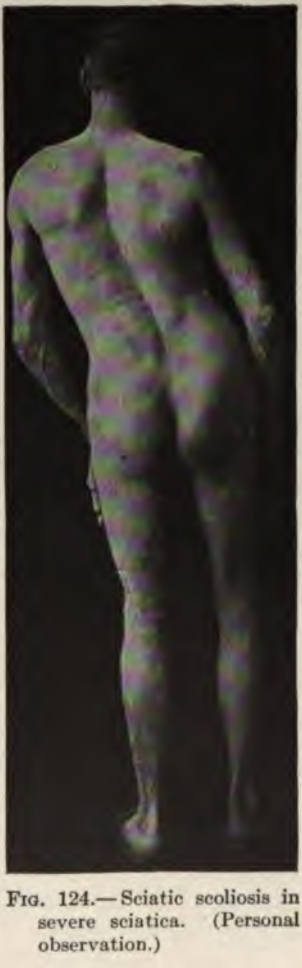

FIG. 124.—Sciatic scoliosis in severe sciatica. (Personal observation.)

The pain usually begins in the lumbar and sacral regions and then gradually passes downward, following the course of the sciatic nerve, through the gluteal region and the posterior surface of the thigh to the hollow of the knee, and then down to the peroneal region (outer part of the leg, outer edge of the foot, and back of the foot), or more rarely to the tibial region (sole of the foot). The accuracy with which many patients point out with the finger the distribution of the pain, corresponding accurately to the anatomical course of the nerve, is a most characteristic feature for the diagnosis of sciatica. Individual cases, however, differ considerably, since at times the pain is felt to a marked degree only in the upper portion and hardly extends at all to the lower leg, while in other cases the severest pain is felt below the knee. This is probably due to the fact that the neuritis, which is the cause of the pain, affects one or another branch of the nerve, either the upper root portion or the terminal branches. The pains themselves are rarely described by the patients as "darting," but are more commonly spoken of as "burning," "boring," etc. The pain is often worse at night than during the day. It is increased by different movements of the leg, by an uncomfortable position, by pressure, by cold, etc. Patients with severe sciatica are therefore almost completely bedridden, and they can take a few unaided steps only with the greatest pain. If we make the patient stand there is often, but of course not always, a very characteristic way of holding the body. As a rule, the trunk is bent over to the opposite side, and the spine thus becomes scoliotic, the convexity being usually toward the healthy side (sciatic scoliosis, see Fig. 124). There has been much discussion about the origin of this scoliosis. It is probably produced entirely by mechanical causes, due to the efforts of the patient to relieve the pressure and to protect the painful nerve as much as possible. For this reason the patient rests on the sound leg in standing, bends the body toward the affected side, and holds the leg slightly flexed, abducted, and rotated outward.

If we examine the affected leg more closely, we shall notice first the sensitiveness of the sciatic nerve to direct pressure. This is often not so great as we might expect, probably because the upper part of the nerve, which is not so easily accessible to pressure, is chiefly affected; but we frequently find the nerve itself quite sensitive to pressure in the middle of the glutæus or at its lower border, in the hollow of the knee, or at the head of the fibula and elsewhere. If the patient be in a lying or sitting position and the thigh be flexed passively, while the lower leg is at the same time kept extended, the procedure gives rise to a gluteal pain which is characteristic, and therefore of diagnostic importance. By this maneuver the sciatic nerve is apparently irritated by being stretched so much (Lasègue's sign). The examiner places his left hand on the knee of the patient, to prevent flexion at the knee joint, grasps the lower leg with the right hand, and then raises the extended leg until the pain is felt. When the knee remains flexed at the same time, flexion of the hip joint is generally not painful. Among the other nervous disturbances we may mention an actual paresis of the muscles supplied by the sciatic nerve (for example, the flexors of the lower leg); this is rare, and, as a rule, indicates more deeply seated disease. The muscular hypertonicity (in part of reflex origin) is more striking; it sometimes takes the form of actual tremor or twitching of the painful leg. A peculiar muscle wave (the so-called "myokymia") is also seen in sciatica. The patellar reflex is usually unchanged; but in severe sciatica we have quite frequently, although, of course, not always, seen the gastrocnemius reflex, obtained by striking the Achilles tendon, absent on the affected side; this is of value in diagnosis. [The patellar reflex is often somewhat exaggerated on the affected side in the early stages of sciatica, and diminished in the later stages.—K.] On careful examination we sometimes find a slight disturbance of sensibility, but it is very rarely marked. In long-continued cases there is almost always a very noticeable atrophy of the affected leg. The circumference of both the thigh and calf is usually several centimeters less on the affected side than on the healthy side. This atrophy is possibly connected with vasomotor disturbances, for the skin of the affected leg often feels decidedly cooler than that of the healthy side. The atrophy, however, is due mainly to the inactivity of the painful leg. [In a considerable number of cases quantitative or qualitative changes in the electrical reactions are found on the affected side. This would indicate that the atrophy was degenerative in character, due to neuritic changes in the nerve.—K.]

Sciatica rarely lasts less than several weeks, and in many cases months or even years may elapse before the pain entirely disappears and the leg completely regains its normal usefulness. The course, however, often shows very marked variations. In simple sciatica (not due to some other underlying disease) the final outcome is usually favorable. We must bear in mind, however, that the disease has a marked tendency to recur, so that any one who has once had sciatica must avoid with special care all exposure which may cause a return of the suffering. The general course of symptomatic sciatica is, of course, dependent mainly upon the nature of the underlying disease.

Diagnosis.—The diagnosis of sciatica is easy in the majority of typical cases, but it may sometimes be quite difficult. It is chiefly confused with lumbago, an acute coxitis, nervous coxalgia (*vide infra*), and psoas abscess. [Osteo-arthritis of the lumbar vertebræ is occasionally mistaken for sciatica. It can usually be distinguished by the spinal rigidity and tenderness.—K.] Lasègue's sign serves principally to differentiate it from a coxitis. Furthermore, we should note that in coxitis abduction of the thigh is painful, which is not the case in sciatica.

Even when the diagnosis of sciatica as such is definite, we must always consider with care the patient's general condition to determine whether we are dealing with a simple primary sciatica (primary sciatic neuritis) or with a symptomatic sciatica following some other underlying disease. In the section on ætiology above we have mentioned the possibilities which the physician must especially bear in mind. In cases where there is a suspicion of simulation (as in accident cases), we must note the accurate localization of the pain and the tender points, and especially the objective symptoms, the almost invariable wasting of the affected leg, and the frequent absence of the Achilles tendon reflex and Lasègue's sign.

Treatment.—In ordinary primary sciatica and in fresh cases we usually begin with "antirheumatic" treatment. The patient should be put to bed and kept warm. We ourselves have treated many recent, as well as more chronic, cases of sciatica with very good results by methodical sweating (hot packs, electric-light baths). Internally, we would first prescribe salicylate of sodium (gr. xxx [gm. 2] t.i.d.), aspirin, antipyrin, salipyrin, or phenacetin, from which we often obtain at least an improvement in the symptoms. Kryofin is recommended as being particularly efficacious in sciatica (gr. vijss. [gm. 0.5] three or four times daily). We have never seen any good effect from oil of turpentine, which is often used in England. "Local derivatives," especially blisters along the nerve, are at present somewhat out of fashion, but we occasionally see decided benefit from blisters, and we therefore still use them quite frequently in obstinate cases. Warm fomentations, a Priessnitz's bandage about the entire leg, etc., usually give relief, and also embrocations with chloroform oil, a spray of chlorid of methyl, etc. If the pain is very severe, so that sleep is constantly disturbed, we must use narcotics, especially subcutaneous injections of morphin. I have not been able to bring myself to employ methodical treatment by morphin injections, as has been recommended from many sides.

Of other methods of treatment besides those already mentioned, we may consider electricity, massage, nerve stretching, and baths, and, above all, certain subcutaneous injections in the region of the trunk of the sciatic nerve.

In electrical treatment we usually employ descending currents of moderate strength, with large electrodes, which we apply to the nerve for five or ten minutes a day, while we include one portion of the nerve after another in the current. Where there is much stiffness in the leg, we open and close the current a few times, in order to excite muscular contraction. Many cases are suitable for the use of the faradic current, especially for the wire brush. Besides electricity, massage and vibratory massage have often given excellent results in sciatica. Details of the technique to be employed may be found in the special treatises on this important method of treatment.

Baths (douches, rubbing) are beneficial in most cases of persistent sciatica. The regular use of simple warm or hot baths (half an hour daily at a temperature of 27° to 28° R. [93° to 95° F.]) is of benefit, especially when combined with massage or electrical treatment. We see still more favorable results at certain bath cures, especially in Wiesbaden and also in Wildbad, Teplitz, etc. Peat baths, and especially hot sand baths (Köstritz, Blasewitz), may have a good effect. In many cases local applications of heat (hot-air apparatuses, hot-air douches, "fango" [volcanic mud] packs, etc.) do good service. On the other hand, many patients report good results from brief rubbing of the affected limb with cold water.

Operative nerve stretching may be tried only in old and very obstinate cases. In some cases it is claimed to have been attended by benefit. We have repeatedly employed bloodless nerve stretching with satisfactory results. This is done by putting the patient on the back and then passively moving the thigh with the lower leg extended for two or three minutes daily as far as possible, and flexing it against the trunk. We have repeatedly employed this method with satisfactory results in the later stages of the disease. The procedure is painful, but appears to be attended with as good results as the passive motions employed in chronic stiffness of joints. [This may be done, if necessary, under ether.—K.]

The method of injection treatment under high pressure, first recommended by Lange, has been used by me repeatedly in my clinic, with very good results. The action of the injected fluid is probably less a chemical one than a mechanical result of the injection of the fluid into the nerve itself and the surrounding tissues (separation of adhesions, etc.). It is therefore of less importance what is injected than how it is injected. Nevertheless, we generally employ the original solution of Lange (beta-eucain, 1 part; sodium chlorid, 8 parts; distilled water, 1,000 parts); this is sterilized by boiling, and on cooling ten drops of a one-per-cent solution of adrenalin hydrochlorate are added. Some physicians use exclusively sterilized isotonic salt solution. For the injection an ordinary aspirating syringe of ʒijss. (10 c.c.) capacity, with a needle three or four inches (8 to 10 cm.) long, is used. Generally the injection is made midway between the tuberosity of the ischium and the great trochanter, the thigh being slightly flexed. The patient lies on his sound side. The site of injection is anæsthetized with ethyl-chlorid, the puncture is then made, and while slowly injecting, the needle is gradually pushed deeper until the region of the nerve trunk is reached. Contact with the nerve, which lies at a depth of about 2½ to 3 inches (6 to 7 cm.) from the skin, is often felt by the patient as a short lightning-like pain. When we think that we have reached the nerve the needle is held still and 2 to 3 or 3½ ounces (60 to 80 or 100 c.c.) of the solution are injected rather quickly and under strong pressure. The needle is then withdrawn and the puncture opening closed with a small piece of plaster. If there is much pain from tension after the injection, this will generally be relieved by moist warm applications.

Exceptionally, a hypodermic injection of morphin is required. In many cases the result of the injection is excellent. We have seen patients with severe sciatica able to walk without any pain a few hours after the injection. Certainly the results are not as brilliant in all cases; then the injections must be repeated every third or fourth day, and in this way a marked improvement or even a cure may be occasionally obtained.

It is very important always to pay attention to the general as well as the local treatment, in order to remove any active causative conditions. A change in the mode of life, avoidance of rheumatic, toxic, and mechanical injuries, regulation of the bowels, etc., are sometimes attended by visible results. While the affected leg must be kept at rest in recent cases, we must urgently insist upon a certain amount of exercise in chronic cases. Any underlying diseases must, of course, be especially treated (iodid of potassium in syphilis, antidiabetic treatment, etc.).

[Mention should be made here of a method of treatment advocated by Weir Mitchell, which is occasionally useful in other forms of neuralgia as well as in sciatica. It consists in absolute rest of the affected limb by application of a splint. With this he combines the application of cold, by an ice bag over the length of the nerve, which may be continued for a number of hours.—K.]

7. NEURALGIA OF THE GENITALS AND THE RECTAL REGION

Neuralgic affections of the parts named are not frequent, but still a number of cases have been described by different observers. Of course most of these observations are of ancient date and many of them are at present perhaps to be explained in some other way.

The pain in these cases has its seat either in the external genitals, or in the urethra, or in the anal and perineal regions. The most frequent form is spermatic neuralgia ("irritable testis" of Astley Cooper), in which there is a most intense pain in the spermatic cord and the testicles, which is almost always associated with an extreme hyperæsthesia of the affected parts. The treatment of this form of neuralgia by narcotics and electricity is often unsuccessful, so that in severe cases resort has sometimes been had even to castration. In women, genuine uterine and ovarian neuralgia does occur, but most cases are either symptomatic neuralgia or a manifestation of hysteria.

Coccygodynia is the name of a form of severe pain in the coccygeal region, seen usually in women, which is much increased by walking, defecation, etc. The affection is so distressing that operations have been repeatedly performed on account of it to remove or to cut around the coccyx. We have twice seen this same symptom as a complication of tabes.

8. ACHILLODYNIA. TALALGIA. TARSALGIA. METATARSALGIA

For practical reasons we will mention briefly here a number of peculiar and often very painful affections of the feet.

Achillodynia is the name given to a pain at the insertion of the Achilles tendon, which is often very obstinate and a hindrance to walking. The trouble often seems to be connected with gonorrhea, in other cases with gout, injury, overexertion, hysteria, etc. We can sometimes make out a slight local swelling, but often there is no objective change. The treatment consists of rest, inunction of mercurial ointment, warm baths and packs, attention to any existing underlying disease, etc.

Pain in the heel is called *talalgia* or *tarsalgia*. Mechanical causes (long-continued standing or walking, the pressure of boots, etc.) often seem to be of significance. Gout is also to be thought of. We must, of course, always consider inflammatory changes, flat foot, etc. Sometimes we cannot discover any cause. Baths, massage, and galvanism are most to be recommended.

Metatarsalgia (*névralgie métatarsienne, Morton's disease*) is an extremely distressing and severe pain, chiefly in the neighborhood of the fourth and fifth metatarso-phalangeal joints. The trouble is observed especially in women in middle life, and it may be sometimes connected with wearing ill-fitting shoes. In other cases injury, gout, etc., are regarded as causes. [It is often due to the pressure upon the nerve between the two phalangeal ends of the metatarsal bones, and it may be relieved by pressure upon the tarsal ends, which causes the phalangeal ends to separate.—K.] The treatment consists of prescribing proper shoes, rest, local fomentations, baths, etc. In some severe cases the painful bones have been resected with success.

There are also other forms of pain in the feet besides those mentioned, which are usually hard to explain. Hysterical conditions certainly play a part in some cases, and are to be taken into account in treatment, but in other cases we often have to deal with mechanical conditions (for example, bearing too great a weight on the feet, as in the obese, continuous standing) and inflammatory conditions which are hard to remove. In all such uncertain cases a Röntgen-ray picture of the bones of the foot is absolutely necessary. It often gives us an entirely unexpected explanation—e. g., fractures of the small bones of the foot which have entirely escaped attention, etc.

9. THE SO-CALLED ARTICULAR NEURALGIAS (Articular Neuroses)

Neuralgia of the joints, first described by the English physician Brodie, was first generally known in Germany after Esmarch proved, by publishing many observations, that apparently severe and very painful diseases of the joints are often found, which are due to no discoverable anatomical disease of the joint, and which we are therefore justified in regarding as nervous affections. Since the localized pain in the joint is the chief symptom in most of the cases of this class, the somewhat unfit term "neuralgia of the joints" has been chosen, although we do not find here, as a rule, such typical and paroxysmal attacks of pain as in genuine neuralgia, and although, too, a number of other symptoms are usually present, which do not occur in genuine neuralgia.

We see neuralgia of the joints chiefly in nervous persons disposed to hysteria, and therefore more frequently in women and girls than in men, but it is also not uncommon in children. We can very often make out a psychical cause for the origin of the disease. The affection, as a rule, is caused by injuries which affect the joint, and which would be in themselves without significance, if they were not associated with a decided fright, and did not direct the patient's thoughts to the affected limb. Articular neuralgias do not, therefore, belong to the pure neuralgias, but to hysteria (traumatic hysteria).

Either immediately after such an occurrence, or often only some weeks later, the patient begins to complain of pain. The knee joint or hip joint is almost always affected, only rarely the joints of the upper extremity. The pain is continuous, but it is more severe at times, especially on motion, or mental excitement. At other times, especially if the patient's attention be diverted from the trouble, it seems to diminish to a considerable extent. It is localized mainly in the joint, but the whole leg is often painful. The patient is usually very sensitive to pressure or jarring, and sometimes we may even discover some painful points on pressure over the joints. The patient cannot walk at all, or at least walking is very painful, and she limps badly. In severe cases, especially if the excessive sympathy of those about her reduces the patient's power to resist her suffering, she becomes completely bedridden for weeks or months. There is usually a decided weakness in the affected leg, which is almost always associated with great muscular rigidity and tension. The leg is extended, flexed, or rotated inward, in just the same way as in genuine coxitis.

The diagnosis of articular neurosis is often quite difficult, but it is almost always possible with long observation of the case. At first, of course, the disease often seems to be a severe affection of the joint, on account of the great pain, the rigid position, and the complete inability to use the leg. The experienced physician, however, is usually struck by the absence of any definite physical changes in the joints, especially of swelling, and also by the changes in the intensity of the complaint, by the influence of mental emotion on the suffering, and finally by the general impression he gets from the patients, the way they behave, and the contrast between their evidently exaggerated complaint and their usual (though, of course, not invariable) good appearance, appetite, and undisturbed sleep. If the patient's attention be diverted, the same pressure on the joint, which before was unendurable, is not even felt. In doubtful cases an examination under chloroform is very advisable. With this, contractures apparently the most severe vanish, and the normal character and mobility of the joint become plainly manifest. A Röntgen-ray examination should never be neglected in severe cases.

As soon as the diagnosis of an articular neurosis is made the treatment has quite definite indications. All embrocations, poultices, bandages, etc., are to be laid aside. The patient is to be brought to the conviction that the pain is dependent only upon the morbid anxiety, and that walking is indeed quite possible if she will only first learn to will to walk again. We therefore make the patient practice walking methodically; these attempts at first prove very unsuccessful and apparently distressing to the patient, but they often lead very speedily to better results. These exercises are very much aided by electrical treatment of the joint, passing a strong current through it or using the faradic brush, and also by local cold douches and massage. Under some circumstances the use of internal remedies may be indicated, in many cases with regard only to the mental condition. We give iron to anæmic patients, and also the nervines. (See the chapters on Hysteria and Traumatic Neuroses.)

10. ACROPARÆSTHESIA

Certain symptoms of sensory irritation, to which F. Schultze has given the name of "acroparæsthesia," have only of late years aroused general attention (Laquer and others), although they have long been familiar to all experienced physicians and have been repeatedly described in medical literature. It is, in fact, quite a common complaint, the chief symptom of which consists of an unpleasant and often even very painful sensation at the tips (*ἄκρα*) of the extremities, especially in the hands, finger-tips, feet, and toes. This sensation is almost constant, or at least it recurs very frequently.

The trouble usually appears in middle life, and it is said to be somewhat more frequent in women than in men. In many cases we can find no special cause, but sometimes we can discover certain injurious influences which have affected the hands or feet, such as past exposure to cold, freezing, much washing in cold water, fatiguing mechanical occupations, chemical irritants, etc.

The abnormal sensations are usually described as darting, prickling, tearing, etc. Their chief seat is, as has been said, in the fingers and finger-tips. Both hands are usually affected, but the trouble is often more pronounced upon one side than upon the other, and certain fingers of the same hand are sometimes more painful than others. The feet (toes) are more rarely affected than the hands; in such cases venous stasis (varicose veins) seems to us to have a certain significance in the ætiology. We must also consider arteriosclerotic changes in the vessels (see page 454). The pain is not equally severe at all times; it is often worse at night and toward morning, ceasing during the day.

If we examine the hands or feet closely we can sometimes detect no objective abnormality, but in many cases certain "vasomotor" phenomena are first of all manifest. The painful parts are cooler and more cyanotic than under normal conditions, but we have also seen very obstinate cases in which the hands usually felt hot and sweated constantly and profusely. The sensibility is normal, or there may be in some parts a very slight blunting of tactile sensibility, or more rarely a marked hyperæsthesia to touch. Motion, as a rule, is undisturbed, but many patients complain of a certain stiffness of the hands. In general, however, it is the pain, often very severe, which limits the usefulness of the hands, and often greatly disturbs the patient's work. There are no special changes in the reflexes. In some cases we have seen very marked trophic changes, especially a manifest smallness and tapering of the terminal phalanges, marked curving of the nails, a shiny appearance of the skin (glossy skin), etc. A short time ago in a very obstinate case, which had lasted for years, we saw torpid ulcers develop on the toes without any external cause; they were very painful and showed no tendency at all to heal.

The course of the disease is in some cases benign, recovery taking place after several months; but in many other cases acroparæsthesia is a very obstinate, chronic condition, lasting for years, and, in spite of its lack of danger, causing the patient great discomfort and distress.

Nothing definite is known as yet as to the nature and special origin of the disease. It is generally assumed that it is an irritative condition of the sensory terminal nerve fibers and terminal apparatus—that is, that it is a purely peripheral trouble; but it is still uncertain whether we have to do with a primary disease of the nerves themselves or with a disease of the smallest terminal arteries and consequent circulatory disturbances with their sequelæ.

The diagnosis of acroparæsthesia is, in general, easy, since the picture of the disease is in fact very characteristic; but we must always bear in mind that such paræsthesiæ sometimes occur as symptoms or precursors of organic central diseases (for example, as precursors of apoplexy, in other brain diseases, in tabes, diabetes, etc.).

Symptomatic treatment is all that is possible, and in this regard the local use of electricity, especially the faradic brush, seems to give the best results, but the constant current, electrical hand baths, etc., are sometimes of benefit. Experience must decide in the individual case as to the influence of thermic procedures (warm and cold hand baths, cold douches, the Scotch douche, etc.). Besides local treatment, we must also pay attention to the patient's general condition (iron, quinin, arsenic in anæmia, etc.) [Drugs which act upon the blood vessels, such as ergot, nitroglycerin, or the nitrites, are sometimes of service.—K.]

CHAPTER V

HABITUAL HEADACHE

(Cephalæa. Cephalalgia)

In addition to the neuralgias, we must speak here of habitual or "nervous" headache, an affection which is very often met with in practice, but in regard to whose precise causes or whose special nature our knowledge is still in many respects very unsatisfactory.

We do not term the symptomatic headaches, so often observed, "nervous headaches." The former come on in acute febrile infectious diseases, in pronounced general anæmia, in the different anatomical diseases of the brain and its membranes, of the skull, the frontal sinuses, etc. Just as little ought we to confuse habitual headache with other painful and well-characterized affections, especially typical neuralgia in the frontal branch of the trigeminus, or in the occipital nerves, or with genuine migraine or hemicrania (*vide infra*). The cases which belong to this class are rather those in which the headache forms to a certain degree a disease in itself, and is the sole, or at least the chief, symptom of which the patient complains, and for which he seeks aid. We know no definite anatomical basis for them. We usually assume disturbances of circulation and of the fine nutrition as the special causes of headache; but the nature of these changes is still wholly unknown. We can also state little that is definite as to the special place where these pains arise. We do not know positively whether painful irritations may arise in the brain substance itself. In case pain should be produced by irritation of the sensory parts of the brain, it would probably not be felt in the head, but it would be projected toward the periphery of the body. The cerebral meninges, however, especially the dura mater, are decidedly sensitive, and hence they may usually be regarded as the special seat of headache. The cranial bones, with the periosteum, and likewise the soft parts (muscles, fasciæ, skin), naturally can also be the real seat of the headache.

The manifold character of the circumstances under which headaches arise renders it probable that the causes of headache are very different in different cases. We have to do either with persons who seem perfectly healthy in other respects, or with weak and anæmic people, or again with robust, very well nourished, "full-blooded" persons with red faces. Hence we look for the cause of the pain, according to the general constitution of the patient, either in an abnormal hyperæmia or in an anæmia of the brain and its membranes—hyperæmic or anæmic cephalalgia. We very often find headache, too, as the chief symptom in nervous, neurasthenic people—neurasthenic cephalalgia. To this class belong especially people who are overworked physically and mentally—scholars, officials, students before an examination, etc. If we believe that we can make out "rheumatic" influences, such as taking cold, or toxic influences, such as alcohol, nicotin, or chronic lead poisoning, we speak of a rheumatic or a toxic cephalalgia; but, strictly speaking, "rheumatic" headache is probably not a nervous headache at all, since it seems to be in many cases, at least, a slight rheumatic inflammation of the external soft parts of the head. Patients with habitual headache often suffer at the same time from chronic gastric disturbances or habitual constipation, so that the latter disorders, in many cases, stand perhaps in a causal relation (toxic influences?) to the headaches. Finally, it is a very important fact that headache is sometimes associated with chronic diseases of neighboring organs, especially of the nose, the nasopharynx, the frontal sinuses, the eyes, and the ear. In very many cases, however, we can find no definite cause at all for the affection, so that we have to do with a genuine idiopathic disease. Habitual headache, existing from childhood, is certainly in most cases the expression of a general "nervous constitution," which is due to congenital conditions which, of course, are still wholly unknown in their details. Habitual headache, therefore, is often an hereditary disease. [One of the frequent causes of persistent headache is eyestrain, due to the persistent unconscious effort of the muscles of accommodation to overcome some error of refraction, such as astigmatism or hypermetropia. The pain is often referred to the frontal region, and it is brought on or made worse by using the eyes for near work. In some cases this eyestrain may be associated with insufficiency of some of the external muscles of the eye. This condition may be associated with normal visual power, so that the patient often maintains that there can be no trouble with the eyes; this is especially true where the refractive error is slight, less than one dioptric. It is therefore essential, in every case of persistent headache, to have a thorough examination of the eyes, in order to determine whether there is astigmatism or hypermetropia. The adjustment of proper glasses, or, in rare cases, tenotomy of some of the external muscles, will often give marked relief, although even in these cases attention must also be paid to the general condition. In many cases the error of refraction is but one out of many causes which give rise to the headache, so that we should never be too confident in our prognosis of cure, and we should also never be content with finding a single cause, but we should search for all possible causes. Referred pains in the head from disease elsewhere have been spoken of on page 165.—K.]

Habitual headache is always a chronic disease. It may last for months or years, or even through the whole life, either being present continually, or, what is more frequent, coming on in separate attacks and lasting several hours or days. These attacks sometimes come without any evident cause, but they may often be referred to definite influences, especially to mental excitement, physical exertion, errors of diet, etc. The pain is felt either in the forehead or in the occiput, or sometimes over the whole head. It is sometimes limited to a definite and quite sharply defined part of the head. The precise form of the pain is described in different ways, either as boring, or tearing, or as if the head were pressed together from without, or as if it would split open. In many cases the intensity of the pain is not great; there is merely a dizziness and a feeling of "pressure" in the head, but in other cases the pain is very severe. In such cases there is also at times a pronounced hyperæsthesia of the skin of the head, so that it may cause pain even to touch the hair.

The general health is almost always disturbed in headache. The patient cannot work, he is often ill-tempered and irritable, and he loses his appetite. We sometimes see more marked gastric symptoms, especially nausea and vomiting (transition forms to migraine, *q. v.*), and sometimes copious perspiration. Severe cases of the disease are of great importance, since by an attack the patient is rendered almost wholly unable to attend to his business. For many women, especially, habitual headache is a factor which dominates their whole life as a restraint and disturbance.

The treatment of headache is a very difficult and usually a thankless task. In the first place, of course, we should try to adapt our treatment to the ætiology of the disease if it is evident. We should therefore never neglect to make a thorough examination of all the organs which are to be considered (nose, ear, stomach, heart, kidneys [eyes], etc.), and pay especial attention to any existing underlying diseases (syphilis, gout, alcoholism, organic nervous disease, etc.) The general physical constitution also requires the greatest attention. For anæmic patients we prescribe iron, arsenic, a country residence, strengthening diet, etc. We order full-blooded persons, especially if they also suffer from indigestion, to follow a restricted diet, to have gymnastic treatment, or to drink bitter waters, or we send them to such health resorts as Marienbad or Carlsbad. Nervous headaches in hysterical and neurasthenic patients require, above all, a rational general treatment: mental treatment, a regulation of their mode of life, cold-water cures, electricity, general faradization, galvanism to the head or to the sympathetic, etc. For persons who suffer from nervous headache in consequence of excessive mental strain and excitement, we most urgently advise complete physical and mental rest for a long time. We send them to the country or make a trial of sea bathing.

The number of symptomatic remedies recommended to relieve headache is very considerable. In most of the tedious cases the patient himself has learned to know his disease perfectly well. Many know that there is no remedy for "their old headaches," and they merely desire to rest, waiting until the pain ceases of itself. Others have become accustomed to employ certain domestic remedies: they put poultices on the head, take a cold or a hot foot bath, put a mustard plaster to the back of the neck, bathe the forehead with cologne water, use a menthol pencil, bind a towel tightly about the head, drink strong tea, smell ammonia or "smelling salts," etc. We sometimes see good results, although frequently we do not, from the internal remedies. These may be used, either during the attack or for a longer period, to prevent the return of the pain. A new remedy often works well, but the benefit is not lasting. There are no special indications for the different remedies, so that we must try to see which one is the most efficacious. Antipyrin is most used of late in doses of 7 to 20 gr. (gm. 0.5 to 1.5). Its efficacy in migraine is undoubted, but it sometimes gives distinct relief in other forms of headache. We may also use aspirin, trigemin, phenacetin, 7 to 15 gr. (gm. 0.5 to 1), and quinin, 5 to 12 gr. (gm. 0.3 to 0.75). In "rheumatic" headaches, which come on after exposure to cold, draughts, etc., we may try sodic salicylate, 0.5 to 1 drachm (gm. 2 to 4). It is of practical importance that a mixture of these remedies (for instance, equal parts of antipyrin and phenacetin) is often more efficient than a single remedy by itself. Of other drugs we may mention paullinia serbilis (guarana, which contains caffein), in 7- to 30-gr. powders (gm. 0.5 to 2), potassium bromid, ½ to 1 drachm (gm. 2 to 4), tinct. gelsemii sempervirens, etc.

Electrical treatment (*vide supra*) has given decidedly good results in many cases, although the mental factor perhaps plays the chief part here. We must always begin electrical treatment with great caution, and try first what method is best borne. Cold-water cures, too, are sometimes beneficial, as we have said above, or residence in the country, at the seashore, or in the mountains.

We can sometimes do the patient good service with the remedies mentioned, but in other cases the evil obstinately defies all attempts at cure. Then, however, the patient has still the encouragement left that the disease often ceases at last spontaneously in advanced age, after lasting years and years.

CHAPTER VI

ANOMALIES OF THE SENSE OF SMELL

ANOMALIES of smell, which depend upon a disease of the olfactory nerve itself, its terminal apparatus, or its central termination, are not infrequently observed, but they have no great practical interest. It is well known that only the upper two turbinated bones and the upper part of the septum of the nares, the olfactory region, are supplied by fibers of the olfactory nerve. The odors excite the special olfactory cells which lie between the epithelial cells. The processes of these cells—that is, the olfactory nerves—pass through the openings of the lamina cribrosa to the olfactory bulbs in which they split up. The ganglion cells of the bulb form neurons of the second order for the olfactory tract. Their nerve fibers pass through the olfactory tract to the brain. The fibers for smell either enter into relation with the optic thalamus or terminate in the cortex of the uncinate gyrus and the cornu Ammonis. A part of the olfactory fibers, however, undergo a decussation in the anterior commissure. The fornix and the corpora albicantia have also relations with the olfactory apparatus, which in man is only a poorly developed organ, while in many animals it has a very decided morphological development.

In testing the sense of smell, we use substances which do not at the same time irritate the sensory fibers of the trigeminus in the nasal cavity. The best substances to use are cologne water, ethereal oils, such as oil of cloves or oil of bergamot, oil of turpentine, camphor, musk, valerian, asafœtida, etc.

Hyperæsthesia of the sense of smell (hyperosmia) makes itself manifest either by a remarkably keen perception of odors, or by an abnormal sensitiveness to them. The latter symptom is often noticed, especially in the hysterical. Patients have headaches, or attacks of fainting, from slight odors, which healthy persons notice but little. Subjective sensations of smell (hallucinations of smell) are quite frequent among the insane, and sometimes during the aura of an epileptic attack.

A diminution of the power of smell (olfactory anæsthesia, anosmia) is not infrequently seen in the different diseases of the nose—coryza, severe inflammation, etc.; also in affections of the base of the skull, such as tumors, and acute and chronic meningitis, which involve the trunk of the olfactory sympathetically, and also in cerebral disease, tumors, etc. In right hemiplegia and aphasia, for example, coexisting anosmia of the left nasal cavity has sometimes been observed. In far-advanced tabes dorsalis we have sometimes found pronounced anosmia, dependent, perhaps, upon an atrophy of the olfactory nerve. Complete or unilateral anosmia, however, is most frequently observed in the hysterical (hysterical hemianæsthesia, traumatic hysteria, etc.). It is important to state that in every marked enfeeblement of the smell the "taste" for many forms of food suffers, since it is well known that their "aroma," as in roast meats, wines, and the different sorts of cheese, is due chiefly to the coexisting sensations of smell.

The treatment of anomalies of smell almost always coincides with the treatment of the primary disease. In case the disturbance of smell makes special interference desirable, we can try electrization of the nasal mucous membrane, or painting it with very irritating or very odorous solutions.

CHAPTER VII

ANOMALIES OF THE SENSE OF TASTE

SENSATIONS of taste are obtained from two nerves—the glosso-pharyngeal and the lingual nerve from the third branch of the trigeminus. The glosso-pharyngeal is the nerve of taste for the posterior third of the tongue and the palate, the lingual for the anterior two thirds of the tongue. The gustatory fibers of the lingual, all, or at least a great part of them, pass over to the chorda tympani, and reach with this the trunk of the facial; they do not remain in the facial, however, as many pathological observations have most plainly shown, but they finally come back to the trigeminus, and, as has been believed up to the present time, probably chiefly by the great superficial petrosal nerve and the Vidian nerve to the spheno-palatine ganglion, in this way passing to the second branch of the trigeminus; or, as is rendered probable by more recent investigations, they pass by means of the otic ganglion to the third branch of the trigeminus and to the Gasserian ganglion.

Finally, all the nerves of taste terminate in the nucleus of the glosso-pharyngeal nerve in the medulla. The further centripetal course of the nerves of taste is not positively known. The chief center for taste sensations is believed to be located in the posterior segment of the gyrus fornicatus.

Hyperæsthesia of taste is rare, and it has been seen almost exclusively in the hysterical. Paræsthesia of taste is sometimes found in patients with facial paralysis, who complain of an abnormal taste in their mouths. In the insane, too, subjective sensations of taste (hallucinations of taste) may occur. Anæsthesia of the gustatory nerves (gustatory anæsthesia, ageusia) is, however, quite frequent. As follows from what has gone before, this may be seen: (1) In affections of the peripheral terminal organs of the gustatory nerves, as in disease of the mucous membrane of the tongue; (2) in affections of the glosso-pharyngeal, such as compression; (3) in affections of the lingual nerve, and of the trigeminus within the cranial cavity; (4) in affections of the chorda tympani, from diseases of the middle ear; (5) in affections of the facial nerve between the entrance of the chorda tympani and the geniculate ganglion; but we know from experience that any obstacle to conduction in this nerve, above or below the points named, causes no disturbance of the sense of taste. Central disturbances of taste are said to have been observed in affections of the posterior portion of the internal capsule. Disturbances of taste are very common in hysteria and allied conditions (traumatic neuroses, etc.).

The test of the sense of taste must be made separately for all the different varieties of the sensation of taste, since partial paralyses of taste are not infrequent. The test is performed by putting small amounts of the substance to be tasted in solution on the tongue with a glass rod or a brush. The anterior and posterior parts are to be tested separately. A solution of quinin or a tincture of nux vomica serves as a test for bitter, a solution of sugar for sweet, vinegar or dilute muriatic acid for sour, and a solution of common salt for salt. We may also use as a test for taste the well-known galvanic taste, which is strongest at the anode, but is also detected at the cathode in even very weak currents, and hence is so often noticed from by-currents in galvanizing the head, neck, etc.

An accurate diagnosis as to the seat and cause of disturbances of taste can be made only by considering the other symptoms which are also present. Direct treatment may best be employed by the aid of electricity.

SECTION II

DISEASES OF THE MOTOR NERVES

CHAPTER I

GENERAL REMARKS UPON THE DISTURBANCES OF MOTILITY

1. PARALYSIS

General Classification of Paralyses.—By "paralysis" we mean the loss of voluntary motion in the muscles of the body controlled by the will. We usually distinguish between the complete loss of the power of active motion (paralysis) and the mere weakening of it (paresis). In complete paralysis of any part of the body, or of a single muscle, the slightest voluntary motion cannot be produced in it; while in paresis of a diseased part certain movements are still possible, but they are more or less below the normal in strength, extent, and duration.

In every portion of the tract that leads from the motor portions of the cortical gray matter of the brain to the muscles—that is, in every part of the so-called great "cortico-muscular conduction path"—disease may lead to paralysis if it takes away from the part affected its power to conduct voluntary motor excitations. Every destruction or inhibition of function of the motor centers in the cerebral cortex, with whose integrity the initiation of voluntary innervation is associated, must also lead to a paralysis in the corresponding muscular region. Finally, diseases of the muscles even may cause paralysis, since the muscles may either have their contractile substance injured, or lose their power to respond by a contraction to any nervous excitation that reaches them ("myopathic paralyses").

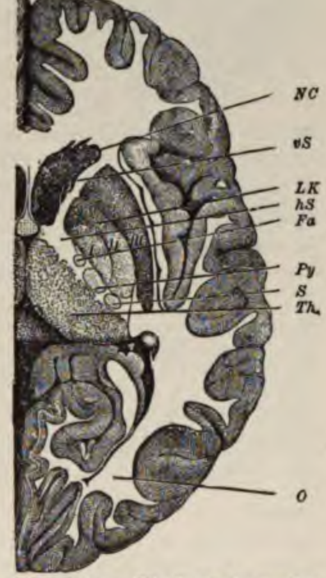

FIG. 125.—Horizontal section through the right cerebral hemisphere. *NC.* Caudate nucleus. *Th.* Optic thalamus. *LK.* Lenticular nucleus (first, second, and third divisions). *vS.* Anterior limb of the internal capsule. *hS.* Posterior limb of the internal capsule. *Fa.* Fibers belonging to the facial nerve. *Py.* Pyramidal tract (motor). *S.* Sensory tract (probably cutaneous nerves and those of special sense). *O.* Occipital lobe.

If we represent to ourselves briefly the precise course of the chief tract for exciting voluntary movements, so far as it is now known, according to all clinical and pathological-anatomical experience, we must put the beginning of this tract, according to all recent experiments, in the region of the central convolutions of the cerebrum and of the paracentral lobule. Here we find the so-called psychomotor centers (see the details in the chapter on Cerebral Localization), from which the motor fibers in the corona radiata converge and pass downward. The latter, after they have united into quite a compact bundle, enter the internal capsule, which they traverse obliquely. As we see in a horizontal section through the cerebral hemispheres (see Fig. 125), the internal capsule consists of two limbs—an anterior, between the lenticular and the caudate nuclei, and a posterior, between the lenticular nucleus and the optic thalamus. The two limbs form an obtuse angle, opening outward, whose apex—that is, the junction of the anterior and posterior limbs of the internal capsule—is termed the "knee of the capsule." The motor tract (*Py*) comprises about the anterior two thirds of the posterior limb of the internal capsule. The fibers for the cranial motor nerves (facial, hypoglossus) lie farther forward than the fibers for the extremities. From the internal capsule the pyramidal tract enters the crusta. It lies first in the third quarter, counting from the inside, then farther down in the middle third of the crusta (see Fig. 126). The fibers for the cranial motor nerves here lie nearer the median line than the fibers for the extremities. Farther downward the tract passes into the anterior half of the pons. In the pons the fibers of the pyramidal tract are somewhat separated, but they come together below it into the compact bundle of the pyramids on the anterior surface of the medulla. The entrance of the motor fibers in the pyramids of the medulla have given the name of pyramidal tracts to their entire extent.

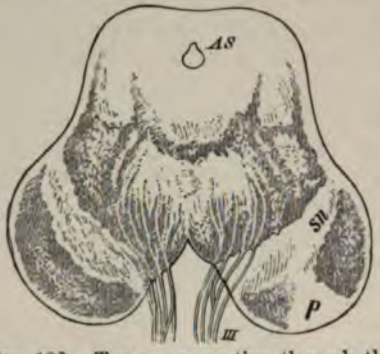

FIG. 126.—Transverse section through the crura cerebri in secondary degeneration of the right pyramidal tract. (From CHARCOT.) *Sn.* Substantia nigra. *P.* The degenerated and therefore translucent pyramidal tract. *III.* Oculomotor nerve. *AS.* Aqueduct of Sylvius.

In the course from the internal capsule to the medulla oblongata all the nerve tracts for the muscles of the head branch off laterally, and, after they have decussated in the median line, they go to the nuclei of their respective nerves (oculomotor nuclei under the aqueduct of Sylvius, facial nuclei in the pons, hypoglossal nuclei in the medulla, etc.). The motor fibers for the trunk and extremities remain in the pyramids, at the lower end of which the decussation of these fibers (motor decussation of the pyramids, *decussatio pyramidum*) takes place. The fibers of each pyramid pass over, for the most part, into the lateral column of the opposite half of the spinal cord, and here form the distinct bundle of the lateral pyramidal tract (*PyS*, see Figs. 127 and 128). A small part of the pyramids, which sometimes seems to be entirely wanting, remains uncrossed, and passes downward as the so-called anterior pyramidal tract (*PyV*, Fig. 127) in the anterior column of the cord on the same side. From the lateral column or the anterior column of the cord, the motor fibers pass into the anterior gray columns of the cord, and are here connected with the large motor ganglion cells of the anterior cornua. We now know that this connection does not

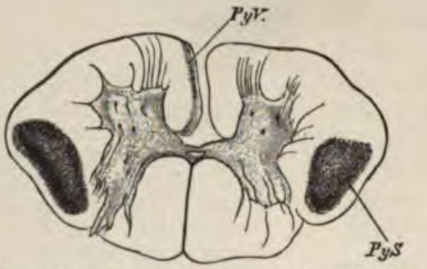

FIG. 127.—Transverse section through the cervical enlargement of the spinal cord. *PyS.* Lateral pyramidal tract. *PyV.* Anterior pyramidal tract (in this case present only on one side).

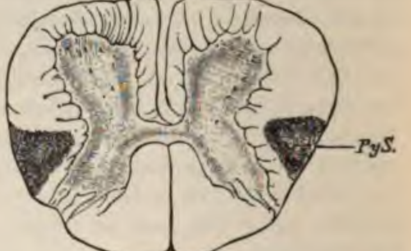

FIG. 128.—Transverse section through the lumbar enlargement. *PyS.* Lateral pyramidal tract. (The anterior tract is no longer present in the lumbar portion of the cord.)

take place in the sense of a direct passage of the lateral tract fibers into the cells of the anterior cornua, but that each fiber breaks up into a "nervous terminal arborization," which surrounds the protoplasmic processes of a ganglion cell of the anterior horn and in this way enters into the closest relation with it. From the ganglion cell of the anterior horn its axis-cylinder process passes off directly as the anterior root fiber, and the anterior spinal roots are further continued as the peripheral motor nerves. In the muscle, as we know, the nerve fiber again breaks up into a terminal arborization ("motor end plate"), which finally transmits the irritation coming from the cerebrum to the contractile muscular fiber.

The long motor tract, just described, the cortico-muscular path or the pyramidal tract, whose course has been quite accurately determined in its details by the results of pathological observations (Türck, Charcot) and investigations into the history of its development (Flechsig), is composed of two neurons connected with each other: the first (central) motor neuron extends from the cell of origin in the cerebral cortex to the terminal arborization of the fiber in the motor nuclei (nuclei of the cranial motor nerves, motor ganglion cells in the anterior cornua); the second (peripheral neuron) extends from the above-mentioned ganglion cells to the muscles, and is accordingly formed essentially by the ganglion cell and the peripheral nerve fiber.

The pyramidal tract, which has just been described, is, however, positively not the only motor tract. It is true that in man it is of the greatest importance, since it conveys all conscious voluntary movements that are executed with a definite purpose. Since movements of this nature (nearly all occupations and manual labors) are performed far more frequently with the arms than with the legs, the part of the pyramidal tract intended for the upper extremity is accordingly much larger than that for the lower. Furthermore, this function of the pyramidal tract explains its smaller development in animals than in man, and its morphological development (myelin-sheath development) occurs comparatively late. In children the pyramidal tract is completed only by the end of the second year; that is, at the time when children begin to execute purposeful movements.

There exists, however, another extremely important and complicated motor function of our body which is concerned with the continued maintenance of our static equilibrium—i. e., the maintenance of the position of the body (lying, sitting, standing) and with movements of propulsion in general (walking, running, jumping). These motor functions can also be consciously and voluntarily influenced from the cortex. For the most part, however, they are executed unconsciously under the control of subcortical centers, which are continuously regulated in a reflex way by sensory impressions. The motor tracts concerned in this function are, according to our present knowledge, the following: (1) The tractus rubro-spinalis arising in the red nucleus of the tegmentum, or Monakow's tract. This passes downward through the tegmentum, and, reaching the crossed lateral columns of the spinal cord, comes to lie anteriorly (ventrally) to the lateral pyramidal tract. (2) Fibers from Deiter's nucleus (vestibular nucleus), which descend without crossing in the anterior part of the lateral tracts and in the anterior tracts. They are known as the tractus vestibulo-spinalis. (3) Fibers from the corpora quadrigemina (tractus tecto-spinalis), which descend in the crossed anterior column of the spinal cord. All these fibers are closely associated with those organs which are intended for the reception of the regulating centripetal stimuli. Monakow's tract is connected by means of the red nucleus and the peduncles with the cerebellum. Its excitation is therefore probably regulated by the impressions received from the muscles, joints, etc. The tractus vestibulo-spinalis is associated with the well-known regulating influence of the labyrinth. For the tractus tecto-spinalis, we may most probably assume regulating influences from the optic nerve or ocular muscles. At any rate we see, therefore, that for the absolutely necessary maintenance of our body equilibrium in standing and in walking, which is a primary requirement and provision for the correct execution of all the finer movements, we have at our command a complete nervous apparatus, the separate parts of which probably can vicariously assume one another's functions. For numerous other motor functions (respiratory movements, swallowing, emptying of the bladder, etc.) there are undoubtedly other separate motor tracts, to which reference will, in part, be made later on.

If we pay attention to the course of the motor tracts described, we shall easily understand certain peculiarities in the distribution of motor paralyses, which are of great importance in diagnosis. Since the motor centers for separate parts of the body, such as the face, the arm, or the leg, are separated from one another in the cerebral cortex, and are distributed over a comparatively large surface, as we shall see more fully later on, it is easily explained why affections of the cortex, if they are not very extensive, may lead to paralysis of only a single part of the body. We call such an isolated paralysis of one part of the body monoplegia, and thus we speak of a cortical facial or brachial monoplegia. Farther downward in the brain, in the internal capsule and the crus cerebri, however, as we have seen, all the motor fibers are collected into one bundle, whose transverse section takes up comparatively little space. Hence we perceive that any disease of the brain, which is situated in this part of the motor tract, may easily make this tract incapable of conduction throughout its whole extent, or at least throughout the greatest part of it. The result must be, then, a more or less complete paralysis of the facial muscles, the arm, and the leg at the same time—that is, of the entire half of the body—a form of paralysis which is termed hemiplegia or unilateral paralysis. We may note here that, as a result of the passage of the motor fibers to the other half of the cord in the decussation of the pyramids and of the other motor decussations, the paralysis must develop on the side of the body opposite to the focus of disease in the brain. Farther down in the spinal cord the fibers coming from both hemispheres, and belonging to the two sides of the body, lie quite near each other. Since many diseases of the cord have a tendency to affect the two halves of the cord at once, or gradually to extend over the whole transverse section of the cord, a simultaneous paralysis of the corresponding portion of the two sides of the body will be apt to arise as a result of this. This form of paralysis we call paraplegia. Diseases in the cervical cord may have as a result a paralysis of all four extremities or of the two arms—cervical, brachial, or superior paraplegia; diseases in the dorsal and lumbar cord a paraplegia of the two legs—inferior paraplegia, often called simply "paraplegia," without further prefix. In diseases of the peripheral nerves we have, of course, again a limitation of the paralysis to the region of the affected nerve. The paralysis may be quite extensive in diseases of a nervous plexus—paralysis of a peripheral plexus; or it may confine itself wholly to the region of a single nerve, or even of a single branch of the nerve—paralysis of a peripheral nerve.

We will have to add many more details to what has just been said, but as a fundamental principle we may now note that hemiplegia is the chief form of cerebral paralysis, while paraplegia is the chief form of spinal paralysis. Monoplegias are usually either cortical cerebral paralyses, or peripheral paralyses.

General Ætiology of Paralyses.—The kind of lesion which leads to paralysis may, in the different cases, be very diverse. From easily understood reasons we can very rarely decide as to the kind of lesion from the intensity and extent of the paralysis, but only from the manifest ætiological factors, from the development and course of the paralysis, from other morbid symptoms that are also present, etc. In general, we may divide paralyses into two groups, according to the nature of their cause—into paralyses from causes that can be discovered anatomically, and into the so-called functional paralyses, in which no anatomical cause for the paralysis can be found; but since our anatomical, and especially our histological, methods of investigation have become better developed and are more employed, the domain of functional paralyses has become gradually more and more restricted, and a definite anatomical cause has now been found for many paralyses which were once regarded as functional.

All diseases of the nervous system may be anatomical causes of paralysis, if they lie in a spot where they damage or destroy the paths of motor conduction. Inflammation, degeneration, new growths, hemorrhages, and severe disturbances of circulation, with their results, especially embolic and thrombotic softening, are found in the brain, the cord, and the peripheral nerves, and under some circumstances give rise to the phenomenon of paralysis. Mechanical lesions of the nervous system also play a great part in the pathogenesis of paralysis, especially injuries, and compression of the brain, the cord, and the peripheral nerves by swellings, new growths, and other diseases in their vicinity.

We also know certain toxic substances which cause direct injury (degeneration) of the motor-nerve territory (chiefly the peripheral motor nerves), and consequent paralysis. Of these toxic paralyses, alcoholic paralysis (*vide infra*) and lead paralysis (*vide infra*) are the most important in their clinical relations; but other poisonous substances, such as copper, arsenic, and certain vegetable alkaloids, may also cause paralysis.

We may group a large number of paralyses together under the term of "paralysis after acute diseases." Since in these cases we always have to do with acute infectious diseases, we may assume, as their most probable cause, certain changes in the nervous system, sometimes in the brain or cord, but much more frequently in the peripheral nerves, which stand in direct relation to the specific infectious material. There seem to be chemical poisons ("ptomaines") which form in the body in infectious disease, and cause degeneration of certain nerve fibers, in the same way, for example, as lead. We most frequently see paralysis appear after diphtheria, diphtheritic paralysis (*vide infra*), or, more rarely, after typhoid fever, smallpox, dysentery, the acute exanthemata, etc. The paralyses which may arise in certain chronic infectious diseases, especially in syphilis and tuberculosis, usually have another origin. Here we usually have to do with the specific morbid product itself (gumma, tubercle), which may invade various parts of the nervous system, but, on the other hand, we must under some circumstances consider the action of toxins even in paralyses in syphilis and tuberculosis. Finally, certain paralyses which occur in certain general constitutional diseases, such as diabetes and gout, are also due probably to toxic influences.

Those paralyses which come on most manifestly because of exposure to cold are termed paralyses from exposure to cold, or "refrigeratory," or frequently "rheumatic" paralyses. Although many spinal diseases, such as myelitis, may perhaps be referred to exposure to wet or cold, we usually reckon among rheumatic paralyses only certain peripheral paralyses, such as that in the domain of the facial nerve. The functional disturbance of the nerves in these cases probably depends upon mild inflammatory changes in them produced by cold, and is accordingly of an anatomical and not merely of a functional nature.

There are, however, quite a comprehensive group of paralyses which we must to-day still term functional paralyses. To this class belong the hysterical paralyses, the paralyses from psychical causes, such as paralysis from fright, the "paralyses from imagination," etc. We shall learn to recognize these more fully in the chapter on hysteria.

In conclusion we must bear in mind the so-called "reflex paralyses," whose ætiology is not yet fully explained—that is, paralyses which may come on in the course of diseases of certain internal organs, especially of the intestine, and of the urinary and sexual organs, as a result of centripetal irritations acting reflexly. An attempt has been made to explain their origin, from analogy with well-known physiological experiments, by the idea that a "reflex inhibition" is excited in certain motor regions by a sensory irritation arising in the diseased organs, a theory which has not yet been fully confirmed. Leyden's hypothesis is scarcely more probable, and it is by no means proved. According to it the paralyses of this class are explained by an ascending neuritis, arising from the organs originally affected (see the chapter on Neuritis). Generally speaking, the whole conception of "reflex paralyses" is still far from clear, and we will do well to be extremely guarded in their diagnosis. Many cases which were formerly regarded as reflex paralyses are to be classed as hysteria, especially as traumatic hysteria (*q. v.*). Lépine has also regarded the paralysis of the arm of the affected side, seen in some cases of empyema, especially after operative interference, as a "reflex paralysis," an explanation which may be proper in some cases, but in regard to which we should be the more cautious, as metastatic abscesses of the brain are not very infrequently found in empyema (see the chapters on Purulent Meningitis and Cerebral Abscess).

General Symptomatology of Paralyses.—We can recognize the existence of a paralysis, independently of the patient's statements as to the impossibility of performing certain motions and functions, only by a careful and thorough physical examination of the power of voluntary motion. This examination in patients with nervous disease must extend to all parts of the body, and demands an accurate knowledge of all the movements that can normally be executed by the different joints, and of the muscles and nerves requisite to produce them (see the tables at the end of this chapter).

In each individual case of paralysis some other symptoms must be considered besides immobility—first, the condition of the paralyzed muscles, and then certain accompanying symptoms that are often present with the paralysis.

In regard to the first point, the trophic condition of the paralyzed muscle is of the greatest diagnostic and practical importance. In comparing a large number of paralyses, a very evident difference in this respect strikes us at once. We see on the one hand paralyses where the paralyzed muscles retain their normal volume for a comparatively long time, and only gradually waste as a consequence of disease and the lack of the normal influences of innervation, although this atrophy never goes beyond a certain degree. On the other hand, however, we also see cases in which there is a considerable atrophy in the paralyzed muscles in a few weeks or months, finally progressing to an almost complete disappearance of the muscles. This distinction is so comprehensive that all the last-named paralyses have been classed together under the name of "atrophic paralyses."

If we once more represent to ourselves the whole course of the motor tracts, from the cerebral cortex to the voluntary muscles, we shall remember that these tracts are composed of two connected, yet independent, neurons. The peripheral neuron directly innervating the muscle has its ganglion cell in the anterior cornu of the spinal cord. It is evident that all higher motor neurons enter finally into relation with the motor cells of the anterior horns, since there is no other communication between the spinal cord and the muscles than that conveyed by the anterior horn cells and the anterior nerve roots. Clinical and anatomical experience teaches us that, in all those paralyses where the cause—that is, the break in conduction of the motor fibers—lies in the first (central) neuron from the cortex, or, possibly, even from more deeply situated centers, up to but excluding the cells in the anterior cornua, there is, as a rule, only a slow and relatively slight amount of atrophy in the paralyzed muscles, while in those paralyses where the cause is situated in the second (peripheral) neuron—that is, in these ganglion cells themselves, or in the motor nerves peripheral to them—a pronounced muscular atrophy rapidly develops. The only possible interpretation of this fact is that the large motor ganglion cells of the anterior cornua have a particular trophic influence on the muscles innervated by them. As we have seen above, the peripheral motor-nerve fiber is nothing but the direct, long cellular process of a motor ganglion cell in the anterior horn. It is therefore easy to understand that this process atrophies when it is separated from the cell body and its cell nucleus, or when the cell itself is destroyed. If, however, the motor nerve degenerates, the muscle belonging to it is entirely separated from all influences of innervation; neither voluntary nor reflex nerve irritations can act upon it any longer. Even the normal muscular tonus, as we shall see later on, ceases entirely. This lack of all functional stimuli apparently causes such a change in the trophic conditions of the paralyzed muscle that its fibers disappear one after the other until finally, in long-continued paralyses, only a slight trace of muscular tissue is left. That this is in reality due to a loss of specific trophic influences of the nerve on the muscle is extremely improbable. In our opinion the atrophy is explained entirely by the complete (even reflex!) inactivity of the muscle when its motor nerve is diseased.

It has been generally believed that the anatomico-histological processes in the rapidly atrophying muscles were also different in diseases of the peripheral motor neurons from the processes of slow and slighter atrophy in paralyses from disease of the central motor neurons. In the first case we were dealing with a so-called "degenerative" atrophy of the fibers—that is, with an atrophy associated with granular and fatty destruction of the fibers—and in the second case only with a "simple" atrophy. Such a histological difference, however, cannot be strictly maintained. We are always dealing, essentially, with a diminution in the size of the fibers, where, however, the transverse striation of the muscle remains plainly visible. We also find a great increase in the nuclei of the muscle, and, finally, we often see an increase of the interstitial connective and fatty tissue.

We observe a further distinction in the condition of the paralyzed muscles if we perform passive motion of the paralyzed parts. There are paralyses where we can perform passive motion of the paralyzed parts at any joint with perfect ease and freedom, and without perceiving the slightest resistance. We term such paralyses "flaccid paralyses," but there are also paralyses where passive motion meets with considerable muscular resistance, so that it can be performed only with a certain greater or less amount of exertion, or not at all, or only within certain limits—spastic paralyses. This distinction is dependent upon the state of muscular tonus in the paralyzed muscles. If this tonus be increased (hypertonia of the muscles), and the muscles be found in a state of marked and permanent contraction, the paralyzed parts show a peculiar stiffness, or even an almost complete spastic rigidity. If the muscular tonus be diminished or wholly lost (hypotonia of the muscles), the paralyzed limbs are flaccid and sometimes even "flapping." Since the muscular tonus is in all probability of reflex origin, it is not remarkable that spastic paralyses are usually associated with an increase, and flaccid paralyses often with a loss of the reflexes, especially the tendon reflexes (*vide infra*). This is not invariably the case, since the reflex paths for muscle tone and tendon reflexes do not appear to be identical. Accordingly, we can readily understand that spastic paralysis can occur only with affections of the central motor neuron (with preservation or even increase of the muscle tonus and reflexes), whereas, all diseases of the peripheral motor neuron, with loss of reflexes and muscle tonus, must produce flaccid paralysis. States of marked permanent shortening of the muscles, whereby certain parts are fixed in an abnormal position, are called muscular contractures. The cause of their development will be spoken of later.

Finally, in every case of paralysis, we must consider the other nervous symptoms which accompany it, since these may also be of great importance in judging of the cause of the paralysis. We must first of all investigate the condition of the reflexes (*vide infra*) in the paralyzed parts, already mentioned, from which many conclusions can be drawn as to the seat of the lesion which causes the paralysis. We must also test the state of the sensibility, both in the skin and in the muscles themselves. Certain attendant trophic and vasomotor symptoms are also to be regarded. The skin over the paralyzed parts sometimes appears cyanotic, or marble-like; it feels cool, is œdematous, and sometimes is peculiarly dry, hard, and scaly. The examination of the electrical excitability of the paralyzed nerves and muscles is of especial importance. The phenomena occurring in these conditions will be particularly described below.

2. SYMPTOMS OF MOTOR IRRITATION

As we have termed the symptoms of motor deficiency "paralysis," we group the symptoms of motor irritation in general together under the name of "spasm." We mean by this all the morbid movements occurring in the muscles involuntarily and even against the will. Although we may find spasm in the smooth muscles, which generally are not controlled by the will, as in spasm of the bronchial muscles, spasm of the vessels, etc., we will concern ourselves here only with the spasmodic movements in the voluntary muscles. We must look for the cause of these in abnormal irritation exerted in some way on the motor tracts, but we know very little of the precise nature and character of this irritation in most cases. The abnormal irritation often acts directly on the motor nervous region, especially in the frequent spasms in affections in the neighborhood of the cortical motor centers; but the motor irritations often seem to be excited secondarily through some reflex channel—reflex spasms.

For a long time two kinds of spasm have been distinguished symptomatically. We term those spasms clonic where the abnormal muscular contraction lasts only a short time, and then is interrupted by a short period of relaxation, to come on again afresh. The affected parts of the body are thus put in a constant convulsive motion. In distinction from this we term those abnormal muscular contractions tonic spasms where the muscle remains spasmodically contracted for a longer time—minutes, hours, or days. The affected part of the body is thus kept motionless in some abnormal position. Both forms of spasm, however, show many transitions and combinations, so that we must often speak of "tonic-clonic" spasms.

A more careful examination of the symptoms of motor irritation, however, gives a still greater number of different forms. We will here group together the most important varieties of morbid involuntary movements.

1. *Epileptiform convulsions* are severe, and usually clonic spasms, at times tonic-clonic, either involving the whole body, and then always associated with disturbances of consciousness, or limited to one half or one portion of the body. By them the whole body or the affected part is put into violent motion, usually thrashing and shaking movements. The pure epileptic spasm in epilepsy is the type of this form of spasm, but precisely analogous spasms, "epileptiform" spasms, are seen in organic diseases of the brain, in uræmia, etc.

2. *Persistent rhythmical contractions* in single groups of muscles are sometimes seen in certain cerebral diseases, such as apoplexy and sclerosis, and also, as we have observed, after acute myelitis. In these the part of the body affected is put in motion by continuous, separate, weaker or stronger thrusts, which follow one another in a regular rhythm. Rhythmical contractions are also seen as precursors or at the end of epileptiform spasms.

3. *Trembling motions*, or *tremor*, as we say in ordinary parlance, are moderate oscillatory motions, which take place from a definite position of mean equilibrium, and rapidly follow one another, with a not very marked excursion.

If the tremor is more pronounced, we term it "shaking." Tremor is an important symptom, almost pathognomonic in many nervous diseases, but we know almost nothing in regard to the more intimate manner of its origin. It probably arises from a central disturbance of the normal fixedly regulated relations of antagonistic muscles. The tremor of paralysis agitans is especially characteristic (*q. v.*). We often see pronounced tremor, especially in the hands, in exophthalmic goiter. We know that tremor is often present in old people—senile tremor; and in alcoholic subjects—alcoholic tremor. Tremor sometimes appears in muscles at rest, that is, not innervated by the will, and sometimes only in those which are moved voluntarily. This latter form of tremor, which is seen most frequently in multiple sclerosis (*vide infra*), is termed "intention tremor." Very marked intention tremor, which is increased by any mental excitement, is also seen as a symptom of chronic mercurial poisoning (mercurial tremor), particularly in mirror-makers, etc.

We may mention here the so-called essential tremor, that is, a condition where the tremor, which is most marked in the hands, is the only morbid symptom and can be referred to no known cause. On energetic voluntary innervation of the muscles the tremor, as a rule, ceases. This form of tremor is sometimes seen in comparatively young people, and even in children. A distinct hereditary predisposition is often present, so that several "tremblers" are found in the same family (hereditary tremor).

We very often find trembling in "nervous" persons, which is at once increased by any emotion (nervous, neurasthenic tremor). Marked tremor is also frequent in hysteria, especially in traumatic hysteria.

4. *Single contractions,* either sudden twitchings, or in the form of a slow contraction of the muscle, are seen with especial frequency in diseases of the cord. They are either single or frequent and persistent. Their mode of origin is not always plain. They may depend on direct motor irritation or they may have a reflex origin. In the latter case each twitching is sometimes associated with a sudden feeling of pain (symptoms of irritation of the posterior roots?). The strongest clonic muscular contractions are seen in a certain group of neuroses, known as myoclonia, electric chorea (*q. v.*), etc., which are evidently closely related to hysteria.

5. *Fascicular and fibrillary muscular contractions* are little contractions in the separate muscular bundles, which may be seen on a close examination of the muscles, but which do not have any special influence on motion. If the fibrillary contractions in a muscle are very pronounced, there may develop an actual "wave" in the muscular substance. We see this symptom especially in atrophied muscles, particularly in spinal progressive muscular atrophy (*vide infra*) and other spinal amyotrophies; for true fibrillary and fascicular muscle contractions are almost always due, in my opinion, to disease and irritation of the motor ganglion cells in the gray anterior horns of the spinal cord. These symptoms of motor irritation of the cord are in a certain sense, therefore, analogous to the epileptiform contractions that result from irritation of the cerebral (cortical) motor ganglion cells. But there also seem to be independent forms of diseases of the muscles, in which we may see for a long time a persistent and extensive muscular wave, associated with pain, profuse sweating, etc.—the so-called *myokymia* of F. Schultze. We must also guard against confusing true fibrillary contractions with a fibrillary muscular

tremor which is not infrequently observed from cold (in people when undressed), or in excited, nervous patients (e. g., accident cases).

6. *Choreic movements* are either slight contractions or quite complicated and extensive, but brief movements, generally not of a clonic character, which usually appear in the face, in one limb, or sometimes over the whole body, without regard to rule. In severe cases they are almost continuous, but in milder cases they are interrupted by shorter or longer pauses. They form the chief symptom of chorea proper, but they are also present in other cerebral affections, such as symptomatic chorea, post-hemiplegic chorea, etc.

7. *Movements of athetosis* is the name we give to peculiar involuntary and usually quite slow movements, which are seen especially in the arms and hands, but also in the head, trunk, etc. The fingers make slow but often very extensive movements, are extended, spread apart, flexed, and moved over and around one another in the most remarkable way. This form of motor irritation occurs as a special disease, "*athetosis*," or as a symptom in certain central nervous diseases, especially the cerebral paralysis of children (*vide infra*).

8. *Coördinated spasms* are symptoms of motor irritation in which the patient performs complicated movements of a convulsive character. Among these are classed certain complicated forms of spasm, such as spasms of jumping, laughing, screaming, etc. They are seen most frequently in severe cases of hysteria, but epilepsy may also occur exceptionally in the form of coördinated spasms.

The so-called forced movements (a compulsory going forward or moving in a circle, rotation about the axis of the body, continuous swaying of the trunk, etc.), which are more continuous in character, belong to the category of the coördinated spasms. Forced movements of this character are seen principally in mental cases and also in hysteria. Certain forced positions of the body are seen rarely in affections of the cerebellar peduncles and of the cerebellum.

9. *Tonic spasm*, as has been said, is the name for all morbid muscular contractions that continue for a long time. Tonic spasm in the muscles of mastication, the masseters, is termed trismus. Tonic spasm in the muscles of the back and neck, by which the whole body is drawn backward, and the vertebral column is bent into an arch with the convexity in front, is called opisthotonos. Tonic rigidity of the whole or a large part of the body is frequently termed tetanus. Tonic spasms are seen not infrequently as an idiopathic condition, but also in tetanus, tetany, hysteria, etc.

10. *Cataleptic rigidity* is the name of that tonic condition of the muscles in which the limbs are deprived of the influence of the will, but are held in position by the muscles in any position given to them passively. This condition is sometimes designated as waxy flexibility (*flexibilitas cerea*). It is seen chiefly in certain cases of hysteria, during the hysterical attack or in hypnosis, but cataleptic states are also present in other cerebral diseases, especially if associated with general mental stupor, such as tumors, meningitis, etc. For certain psychoses (catatonia), the occurrence of cataleptic symptoms is very characteristic. We should like to mention here that in children one or two years old, suffering from any other kind of trouble, marked catalepsy has not been very infrequently observed. This all proves that catalepsy is always

psychical in origin. It depends on a psychical interference with the normal voluntary muscular innervation, and is therefore always associated with a certain degree of stupor. (See the chapter on Hysteria.)

11. *Associated movements* are abnormal movements which appear, while making voluntary movements, in muscles which have no connection with the movement willed. Thus in hemiplegia associated movements sometimes take place in the arm when the patient wills to move the leg alone. In spinal cases the movement of one leg is sometimes accompanied also by an unintentional movement of the other leg. Associated movements are commonest in the muscles of the same limb. Thus we often see in hemiplegia or in spastic spinal paralysis that patients cannot flex the leg up toward the body without at the same time producing a marked dorsal extension of the foot as an associated movement (so-called tibialis phenomenon). In old peripheral facial paralysis (*q. v.*) we very often see associated movements in the facial muscles.

Besides the conditions of motor irritation, other attendant nervous symptoms often occur at the same time. Symptoms of motor paralysis and irritation are very often combined with each other, since the different forms of spasm may appear not only in groups of muscles whose motion is otherwise normal, but also in paretic or paralyzed muscles. In general convulsions the state of the consciousness deserves special attention. Genuine general epileptic attacks are associated with complete loss of consciousness, hysterical attacks are often associated with clouding of consciousness, but in most of the other forms of spasm the consciousness is unaffected. Finally, it is worthy of mention that tonic spasms especially are attended by a feeling of decided pain, which is probably due to an irritation of the intramuscular sensory nerves. Such painful tonic muscular contractions are termed cramps. Among them are the well-known painful spasms in the calves, etc.

3. ATAXIA

In executing all normal complicated movements we need the simultaneous action of several muscles. Consider the numerous muscles which must be put in activity in walking, in grasping, and in all the manifold employments of the hands. Hence, in order to perform such movements correctly, it is not only necessary that all the muscles concerned should be innervated by the will—that is, that they be not paralyzed—but that we should also be able so to modify the innervation of each individual muscle that its contraction corresponds precisely to the special part of the work belonging to it. A prescribed voluntary motion can take place only when, first, all the muscles requisite for it come into no less, but also no greater, action; second, when each individual muscle contracts only so far and so strongly as its special task requires; and, third, when the conditions in the time of innervation take their normal course—that is, when all the muscles involved contract at the same time or in the proper order one after another. A movement which is executed in such a prescribed manner we call a coördinated movement, and the process of modifying properly the innervation of the different muscles necessary for a complicated movement we call the coördination of motion. We must especially bear in mind that the simultaneous action of several muscles is so far necessary, even for what seem to be the simplest movements, that muscles antagonistic to those moved must also come into activity. Only by the aid of the ever-ready antagonistic muscles can we grade our movements as finely, or check or hasten them as rapidly, as is demanded for the execution of almost all voluntary movements. We must bear in mind, furthermore, that in addition to the muscles which are required to execute a definite motion, numerous other muscles must act simultaneously to furnish a fixed point of support to those muscles which are actually in action. Thus, for example, to execute most of the motions of the hand with precision, it is necessary that the entire arm be held at the same time in the correct way. In short, all our motions require the extremely intricate action of entire groups of muscles.

Nervous pathology is rich in facts which can make the idea and the necessity of the coördination of motion clear to us. We often see disturbances of motility which make the patient incapable of any fine motor acts, and yet which do not depend at all upon any motor weakness or paralysis, but only upon a disturbance in the coördination of motion. Such a disturbance we call ataxia, and we speak of an ataxia of the arms, of the legs, etc., when the parts named can perform all the motions and often even retain their full strength, but these movements show, usually at once, a striking, disordered, uncertain, "ataxic" character.

Many theories have been advanced as to the precise cause of ataxia. In general, however, there can no longer be a doubt that ataxia depends, at least in many cases, upon a disturbance of centripetal influences. We must suppose that our movements are constantly kept in order and control (coördinated) by centripetal excitations coming from the skin, but principally from the muscles themselves, the fasciæ, tendons, and joints, although they are not all by any means consciously perceived. By such excitations the child learns gradually to change his original uncontrolled and unregulated movements into orderly movements. In the adult most movements have already been so long practiced as to be sure and steady. The learning of a new movement, a new dexterity, however, takes place in precisely the same way. Even a long-practiced movement (walking, grasping) needs the constant control and regulation exerted by centripetal excitations. Every deviation from the normal course of a coördinated movement produces at once a corresponding peripheral excitation which passes centripetally to the proper motor apparatus, and gives rise there immediately to the necessary correction of movement. In this way our movements (provided the nervous system is intact) always adapt themselves to external conditions, and appear, therefore, always regular and purposeful. Even coarse external interferences (e. g., stumbling, etc.) are immediately compensated unconsciously by centripetal (i. e., reflex) regulation of the movements. If, on the other hand, these controlling influences are lacking by reason of any disease of their corresponding centripetal tracts, ataxia is the necessary result. The movements then become swaying and uncertain. A definite movement can be executed only in a roundabout and laborious manner. The large coördinating apparatuses for standing and walking also lose their certainty of action; the gait becomes swaying and clumsy, with the legs wide apart.

In very many cases the anatomical basis of the ataxia can be easily proved to be a disease of the centripetal nerve tracts. Above all, it is the degeneration of the sensory fibers coming from the deeper parts (muscles, fasciæ, joints) that constantly leads to ataxic disturbances of motion. Since these fibers, as mentioned above (*vide* page 156), pass through the white posterior columns of the cord, disease of the posterior columns (tabes, Friedreich's disease, posterior myelitis, etc.) are often accompanied by ataxia. But ataxia also follows diseases of the corresponding peripheral nerves (in some forms of polyneuritis) as well as diseases of the centripetal prolongation of the fibers of the posterior columns into the lemniscus. From this it follows that the place where the centripetal stimuli are regulated is not located in the spinal cord, but in the brain, the control of the finer movements of the extremities being probably limited chiefly to its motor cortex. Hence, we occasionally see ataxia in affections of the motor cortex of the brain. On the other hand, the centripetal regulation of trunk movements in standing and walking is located chiefly in the cerebellum. Disturbances of the cerebellum are therefore frequently associated with an uncertain and swaying gait (cerebellar ataxia), while the execution of individual motions of the arms and legs is performed in a perfectly coördinated manner. The question has been much discussed whether ataxia, in consequence of the loss of centripetal stimuli, is necessarily always associated with a corresponding loss of sensation. It may be stated as a general rule that organic disturbances of the muscular sense are always associated with ataxia. On the other hand, ataxia may also be present without marked disturbance of the conscious muscular sense, since the degree of an ataxia and that of a possible coexistent disturbance of the deep sensibility (muscle sense, etc.) are not parallel. The regulation of the voluntary motions through centripetal stimuli occurs, therefore, through tracts which are not entirely identical with those conveying conscious muscular sensations. We will refer later on repeatedly to these difficult and interesting problems (compare chapters on Multiple Neuritis, Tabes, Multiple Sclerosis, etc.).

It is not easy to answer the question, whether every ataxia is due to a loss of centripetal stimuli. It seems entirely possible that, in spite of normal centripetal regulating influences, disturbances could occur in the motor centers, or even possibly in the motor tracts themselves—e. g., differences in the rapidity of conduction of stimuli, which would prevent a normal coördinated movement. We would then be dealing not with a "sensory," but with a "motor ataxia." For example, the ataxic disturbances of motion seen in multiple sclerosis (*q. v.*) are perhaps purely motor.

To test ataxia of the arms and hands we have the patient execute certain movements of precision, such as touching an object held in front of him, or touching a definite part of the body with the tip of the finger, placing the finger-tips together, or performing more complicated movements, such, for example, as are required in writing, tracing of definite lines, tying a bow, buttoning, etc. Ataxia of the legs is best tested by the so-called knee-heel test—that is, placing the heel of one foot upon the knee of the other leg. It is a good plan to mark several points on one leg with chalk, and have each one of these accurately touched with the other heel. Ataxia of the trunk is tested by observing the gait along a prescribed line, standing with feet closely approximated, walking upstairs, etc. In every ataxia due to the loss of centripetal stimuli, the patients attempt to replace the muscle sensations by eye control—that is, by the control of visual sensations. Hence, the ataxia becomes more evident when the eyes are closed. Especially often we see that while the patient can stand fairly well with the feet closely approximated while the eyes are open, marked swaying of the entire body ensues when the eyes are closed (so-called Romberg's sign). In ataxia with loss of muscular sense in the arms, the patient with his eyes closed is unable to touch a point, the location of which was previously known to him, or else is very uncertain in touching it.

4. GENERAL REMARKS UPON TESTING THE REFLEXES AND UPON THE CONDITION OF THEM

In any case of nervous disease we should never neglect to test the reflexes, on account of their great diagnostic importance. The two chief groups of reflexes, the cutaneous reflexes and the "tendon reflexes," must be distinguished from each other.

Cutaneous Reflexes.—We term the muscular contractions, excited reflexly by irritation of the sensory centripetal cutaneous nerves, cutaneous reflexes. These are usually present only to a slight degree in the upper extremities; but we can sometimes excite reflexes even here by pricking or pinching the skin, especially the skin of the fingers. The very marked reflex in many people caused by tickling the axilla is well known. The test of the cutaneous reflexes in the lower extremities is much more important. The soles of the feet are the most sensitive parts for exciting a reflex. Simply tickling the soles with the finger is a sufficient irritation (the tickling reflex), and so is the prick of a pin (the prick reflex), or stroking the skin hard with a blunt object, usually the handle of a percussion hammer (the stroking reflex). Thermal irritants are also very suitable for exciting a reflex, especially bits of ice held to the skin (cold reflex). It is often advisable to try all these methods, since with diminished reflex irritability a reflex contraction in the leg can often be excited by some one of them alone. We should also examine the reflex irritability of the rest of the skin, as well as the soles of the feet, by a pinprick, by pinching a fold of the skin, etc. We should especially remember that in nervous diseases there is often a delay in the reflex, so that the reflex contraction appears only when the irritation has lasted for a certain time. Thus in many diseases of the cord, as we have often seen, the reflex follows only after we have kept a needle sticking into the skin for some time or have pinched a fold of skin continuously for several (ten or fifteen) seconds, a phenomenon which has a connection with the fact of the "time summation of reflex irritation" known from physiology. The summation of reflex irritation not infrequently coincides with the summation of irritation from the sensation of pain (*vide supra*, page 151), so that, after the irritation has acted for a certain length of time, the sensation of pain and the reflex contraction occur at the same time. In the stroking reflex (a rapid, vigorous stroking of the sole of the foot or of other parts of the skin with the handle of a percussion hammer) we have a local summation of reflex irritation, since many different parts of the skin are rapidly irritated one after another. In the tickling reflex the local and the time summation of reflex irritation are united. All parts of the skin do not show the same "reflex sensibility." The reflexes are usually set free more readily from the sole of the foot than from the skin of the leg or thigh. The reflex contraction in such cases consists usually of a dorsal extension of the foot and a coincident flexion of the knee and hip. After irritating the skin of the thigh we sometimes see reflex contractions in the extensors of the lower leg. The stronger and more persistent the irritation, the more energetic and widespread is the reflex contraction. Where the reflex irritability is actively increased, sticking a needle into the sole will often produce a vigorous flexor contraction in the irritated leg, and also a weaker contraction at the same time in the other leg.

Of special practical importance is an accurate observation of the reflex movements of the toes, particularly of the great toe. These reflexes are best tested by firmly grasping the foot to be examined in the left hand, and then with either a pin or the handle of the percussion hammer making light and strong strokes along the sole of the foot over the ball of the toe, the inner border of the foot, and other places. In healthy people the reflex movement of the toes almost invariably occurs as a plantar flexion, while, on the other hand, in many diseases of the nervous system a dorsal extension of the toes and particularly of the great toe occurs (Babinski's toe reflex). This reflex dorsal extension of the great toe indicates, with almost absolute certainty, organic disease of the motor pyramidal tract, and we shall therefore have frequent occasion to refer to this phenomenon later on. It is an interesting fact that in newly born infants with still undeveloped pyramidal tracts, the cutaneous reflex of the great toe also occurs as a dorsal extension.

[Oppenheim has shown that a similar dorsal extension of the great toe may be produced by drawing some blunt instrument, such as the handle of a percussion hammer, downward along the inner border of the tibia, exerting moderate pressure while so doing. This reflex is of equal value and of the same significance as the Babinski reflex, and is sometimes obtained when the Babinski reflex cannot be elicited. In both these reflexes the great toe is extended slowly, which distinguishes them from the quick extension of the toe when the foot is withdrawn semivoluntarily from the irritation. We seldom fail to obtain some form of plantar reflex, which is alike on the two sides, but sometimes, instead of the normal flexion of the toes, there is a contraction of the tensor vaginæ femoris (Brissaud's reflex) which may be overlooked. Occasionally there is a spreading of the toes, like a fan, which is of no special significance.—K.]

We must also mention two special forms of cutaneous reflex which are often examined on account of their diagnostic importance: the abdominal reflex and the cremaster reflex. The abdominal reflex is best excited by stroking the abdominal wall rapidly and lightly with the handle of a percussion hammer or with the point of a needle. If we stroke from without inward on the linea alba, we can distinguish, according to the level of the irritation, an upper, middle, and lower abdominal reflex. On stroking from above downward, these contractions unite. In health the abdominal reflex is only very rarely absent, chiefly in persons with a very lax abdominal wall and in the corpulent. In cerebral hemiplegia the absence of the reflex on the paralyzed side is an important sign. In spinal diseases the reflex may be absent on one or both sides, which should lead us to bear in mind that the centripetal portion of the reflex arc of the lower and middle abdominal reflex enters the spinal cord through the posterior roots of the tenth to the twelfth intercostal nerves, and of the upper abdominal [epigastric] reflex through the posterior roots of the ninth intercostal nerve (Dinkler). The cremaster reflex is a reflex ascent of the testicle when the inner side of the thigh is forcibly stroked or when sudden deep pressure is exerted by the hand a hand's breadth above the internal condyle. This reflex is almost always present in healthy boys and men, although in varying strength. The clinical significance of the cremaster reflex is similar to that of the abdominal reflex. The reflex arc of the cremaster reflex is at the level of the first and second lumbar nerves. But this reflex may also be elicited over greater cutaneous areas (as far as the lower leg). In these and other reflexes we are therefore able in every case to determine the area of the reflex zone. We must take care not to confuse the cremaster reflex with the contraction of the dartos, which can also be elicited by stimulation of the skin of the scrotum or of the anal region (so-called scrotal reflex). Other cutaneous reflexes—the gluteal, the mammillary, etc. —have at present no special practical significance.

Tendon Reflexes.—Of almost greater practical importance than the investigation of the cutaneous reflexes is the test of the phenomena classed under the name of the "tendon reflexes," and first carefully investigated and described by Erb and Westphal in the year 1875. We understand by tendon reflex those muscular contractions which arise from the brief mechanical irritation of the tendons and analogous parts, such as the periosteum and fasciæ. Westphal originally held that these contractions were the result of direct mechanical irritation, but later accurate clinical and experimental work has shown that the "tendon reflexes," as Erb had maintained from the beginning, are true reflex phenomena, starting from the mechanical irritation of the sensory nerves in the tendons, periosteum, etc. The tendon reflex of most practical importance is the patellar reflex, "knee phenomenon" (Westphal) [or "knee-jerk" (Mitchell)]—that is, a contraction occurring in the quadriceps after mechanical irritation of the ligamentum patellæ by a quick blow with a percussion hammer. In order to evoke this reflex it is especially necessary for the person examined to avoid all active muscular tension in the leg, especially in the extensor. If we examine patients who are not in bed, we can test the patellar reflex by making the patient, while sitting, cross the leg to be tested over the other, and by striking the patellar tendon as the leg hangs loose; but it seems to us more convenient to direct the patient to extend the leg until it forms an obtuse angle with the thigh with the opening of the angle below. If the sole be set fully on the floor the extensor cruris is made tense in this position, and we can very easily and plainly excite the contraction of the quadriceps by striking the patellar tendon. [It is often better to have the patient sit on a high stool with the legs relaxed and the feet clear of the floor. The legs should be bare, without any constriction.—K.] When the patient is in bed, it is best to raise the leg to be tested into a semiflexed position by putting the left hand under the hollow of the knee; the foot remains lying on the bed, and the patient should avoid any active muscular tension. Or we have the patient cross the leg we wish to examine over the other, which should cause it to assume a relaxed and slightly flexed position. In all doubtful cases it is absolutely necessary to test the patellar reflex in various ways. If the patellar reflex be weak and hard to detect, we should try the proceeding advised by Jendrássik, which consists in testing the reflex while the patient grips the two hands together, and tries with all his strength to pull them apart. By this or any other vigorous tension of the muscles of the upper extremities—e. g., squeezing the left hand of the observer firmly, which is also a good method—the muscles of the legs are relaxed, while any voluntary innervation is avoided, and thus is explained the increase of the reflex, which is often pronounced. If by the help of this "Jendrássik's test," in spite of repeated attempts in different positions of the patient (sitting and lying down), no reflex is to be elicited, we may regard it as absent, a condition which is almost always of pathological significance, since complete absence of the patellar reflex practically never occurs in health. The reflex arc in the cord for the patellar reflex is found in the second lumbar segment.

The second important tendon reflex to be provoked in the lower extremity is the Achilles tendon reflex [ankle jerk]. If we give to the foot of the person examined (who should lie on his side in bed with slightly flexed knees, or kneel on a chair) a passive position of slight dorsal extension, so that the tendo Achillis is a little tense, and then strike the tendon a quick blow, a marked contraction of the gastrocnemius follows. Precisely the same reflex is usually to be elicited by striking the plantar fascia. The Achilles tendon reflex is almost as constant in healthy persons as the patellar reflex. Its reflex arc in the cord lies at the level of the third to the fifth sacral segments. Where the tendon reflexes are abnormally increased, however, the Achilles tendon reflex is very vigorous, and then we can very often produce it in the following especially characteristic manner. If we make a sudden, short, vigorous, passive dorsal extension of the foot, the tendo Achillis is suddenly made tense, and thus is irritated mechanically. As a result of this, there is a reflex plantar flexion of the foot. If now, by persistent passive dorsal extension of the foot, the tendo Achillis is again made tense, there follow by turns new plantar and dorsal movements of the foot, so that the foot is thus put into a vigorous tremor. This symptom, which can only exceptionally be provoked in healthy persons, is termed ankle clonus (foot clonus), or "foot phenomenon" (Westphal). Where there is a very great increase of the tendon reflexes the tremor is sometimes not confined to the foot, but the whole leg falls into a vigorous clonus, a symptom which was once given the unsuitable name of spinal epilepsy, since it apparently came on spontaneously as a result of some tension of the tendons. We can also obtain the patellar reflex in the form of a clonus if we pull the patella firmly down with the finger and force it downward by a sudden blow on the finger.

The two symptoms described—the patellar reflex, and the Achilles tendon reflex or foot phenomenon—are practically the most important, and are the most often tested, but they are by no means the only reflexes in the lower extremity. Besides the reflexes from the special tendons, we also frequently obtain muscular contractions by striking the periosteum and the fasciæ, which have been termed the periosteal and fascia reflexes. Thus a contraction in the quadriceps often follows a blow on the anterior surface of the tibia when the reflex excitability is increased. We can designate this also as an increase of the reflex zone, a circumstance which almost invariably indicates an increase of the patellar reflex. We also see contractions frequently in the adductors of the thigh on striking the internal condyle of the femur, contractions in the muscles of the posterior aspect of the thigh in striking the calves, etc.

Under normal conditions the tendon reflexes in the upper extremities are sometimes quite weak or entirely absent, but where the irritability is abnormally increased we see even here the most various and vigorous reflexes. The most important and most constant are the periosteal reflexes, from striking on the lower end of the radius and ulna. As a rule, in our observations, we have usually obtained a contraction in the supinator longus and biceps from a blow on the head of the radius, and a weaker contraction in the same muscles from the head of the ulna, and also, very frequently, pronation of the forearm and flexion of the wrist and fingers. The deltoid also contracts not infrequently from a blow on the lower end of the ulna. Direct tendon reflexes in the biceps and triceps on striking their own tendons are also almost always to be obtained. [The triceps reflex seems the most constant of all the reflexes of the upper extremity.—K.] We may also not infrequently obtain a contraction in the biceps by a blow on the clavicle. A persistent clonus in the hand upon passive volar flexion sometimes occurs, but it is rare.

A more detailed account of the behavior of the cutaneous and tendon reflexes in the individual diseases of the nervous system will be given later in the special chapters, where we shall also learn the present theories as to the course of the reflex tracts and the influence of other nerve tracts upon them.

[**Muscular Tonus.**—Changes in the muscular tonus are often of considerable importance (A. Knapp). If the extended leg be flexed upon the trunk, while the patient is lying on his back, it cannot ordinarily be raised beyond the vertical position. If the leg be now flexed at the knee, the thigh can be flexed still more, showing that the obstacle is in the tension of the muscles and not in the joint itself. The tonus may be tested in this way, and also by making various passive movements of the limbs. It is an involuntary reflex contraction of antagonists from the sensory irritation of passive motion. A diminished muscular tonus (hypotonia, atony) is usually found in conditions attended with diminution of the tendon reflexes, and an increased tonus (hypertonia) in conditions attended with exaggerated tendon reflexes. This is not invariably the case, for hypertonia is not found in cases of neurasthenia, where the reflexes are often much increased. Hypotonia is common in neuritis, tabes, and the various forms of muscular atrophy, in some cases of cerebellar disease, rarely in hemiplegia, after a convulsive attack, in epilepsy, hysteria and chorea, in catatonic states, and in general paralysis. In conditions of hypertonia it is not possible to raise the extended leg nearly to the vertical position, and there is a general resistance and rigidity of the affected limb on passive motion. Hypertonia is one of the early signs of disturbance of the pyramidal tract and of all spastic conditions. In meningitis, if an attempt be made to flex the extended leg on the trunk, the procedure is painful and the lower leg is promptly flexed by a reflex contraction (Kernig's sign). This is not a true hypertonia, but a reflex contraction due to the pain caused by stretching the nerves.—K.]

Mechanical Muscular Irritability and Paradoxical Contraction.—"*Direct mechanical irritability of the muscles*" is shown by the occurrence of energetic brief contractions of single muscular bundles or of the whole muscle from a direct blow on the belly of the muscle with a percussion hammer. In such cases, however, we are dealing, in my opinion, not with a direct muscular irritation, but with a mechanical irritation of the nerves which enter the muscle, since the muscular contractions are always strongest when we strike the corresponding point of entrance of the nerves, precisely the same as faradic irritation of the muscle. The ensuing muscular contraction, however, may sometimes be a reflex, evoked by mechanical irritation of the fascia stretched over the muscle. These contractions just described are to be distinguished from the so-called idiomuscular contractions. We see these most plainly if we give a vigorous blow with the ulnar side of the hand or with a percussion hammer to the belly of a muscle, such as the biceps. A small transverse, circumscribed muscular swelling forms at the point struck, and gradually disappears again. The test for mechanical muscular irritability has not yet attained any special practical importance.

"*Paradoxical contraction*" is the name which Westphal has given to a symptom seen especially in the tibialis anticus, and rarely also in the flexors of the leg and forearm. It is when the foot, after being put in passive dorsal extension, remains in this position even after the expiration of a considerable time (several minutes), and a marked prominence of the tendon of the tibialis anticus is usually visible. We cannot at the present time give a definite explanation of this phenomenon, which so far has been observed, but on the whole only rarely marked, in various spinal and cerebral diseases, multiple sclerosis, paralysis agitans, etc.

5. GENERAL REMARKS UPON THE CHANGES OF ELECTRICAL EXCITABILITY IN THE MOTOR NERVES AND MUSCLES[1]

Electricity, since the investigations of Duchenne, Remak, Benedikt, Moritz Meyer, von Ziemssen, Brenner, Erb, and others, has become not only one of the most prominent therapeutic aids in the treatment of nervous diseases, but it also plays an important part in the examination of nervous patients, since the test of the electrical excitability of diseased nerves and muscles gives us a large amount of valuable information in regard to diagnosis and prognosis.

Every complete electrical examination must be made with both currents—the faradic or induction current (usually the secondary current) and the galvanic or constant current. One "indifferent" pole is usually put on the sternum or the back of the neck, and the other "testing" pole on the nerve or muscle to be tested. For the indifferent pole we use a large electrode about 12 cm. long), and as a testing pole a smaller electrode, the best being Erb's "normal electrode," with a surface of 10 sq. cm. The excitement of the muscle from the nerve is called indirect; the excitement from placing the electrode on the muscle itself (where, of course, the excitement of the intramuscular nerves cannot be excluded) is called direct. Those points on the human body where the different nerves and muscles are most easily accessible to the electrical excitement are to be found in Figs. 129 to 134, taken from Erb's handbook.

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[1] In regard to all the details of electrical diagnosis and electro-therapeutics we would refer to Erb's "Handbuch der Elektrotherapie." Leipsic, Vogel, 1886. [Translated by De Watteville. New York: Wm. Wood & Co., 1887.] E. Remak's "Grundriss der Elektrodiagnostik und Elektrotherapie." Vienna, 2d ed., 1909, etc. [Turner, "A Manual of Practical Medical Electricity." London, 1904.]

In faradic examination the normal condition is that we can provoke marked muscular contractions both from the nerves and from direct excitement of the muscles at the points generally accessible to excitement. As von Ziemssen has found, the points at which the muscle can most easily be made to contract by "direct" excitement correspond exactly to the points where the motor nerves enter the muscle. The direct faradic muscular excitation is, therefore, always a nerve excitation. In order to determine the excitability quantitatively we measure the distance between the two coils of the induction apparatus (coil distance) at which the first minimal contraction of the muscle occurs. On increasing the current, the minimal contraction gradually passes into a vigorous tetanic contraction of the muscles.

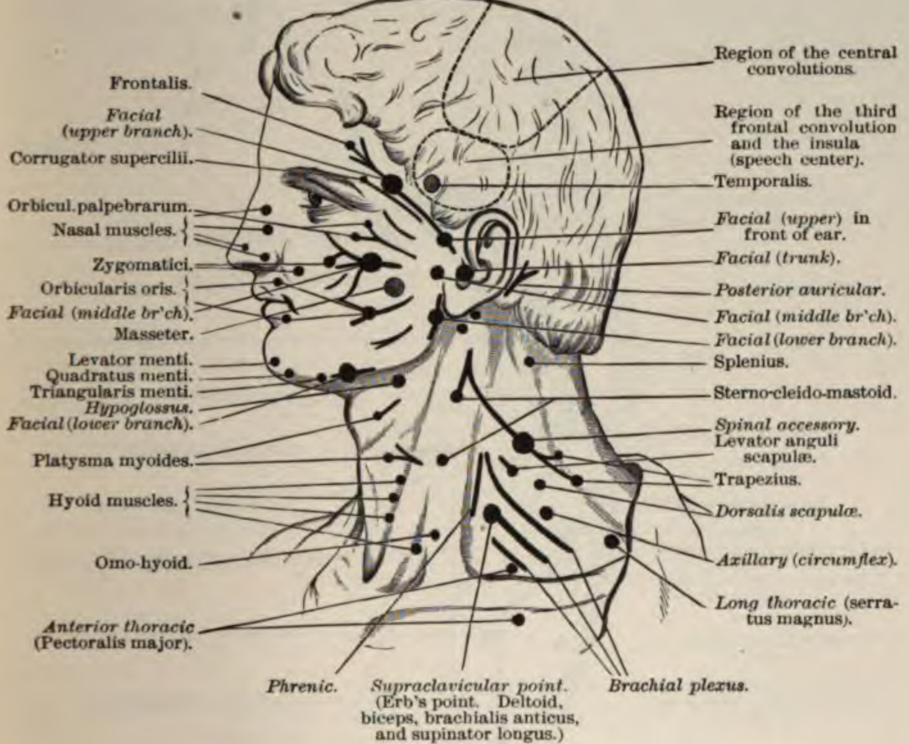

FIG. 129.

Galvanic examination is performed by the aid of a "current reverser," by which the current at any desired strength can be rapidly opened and closed and the testing pole can be made either the negative pole (the cathode or zinc pole) or the positive pole (the anode or copper or carbon pole).

The nerves and muscles are excited not by the continuous flow of the electrical current, but only by its variations and the sudden opening and closing of the current.

By the help of this "polar method of investigation" (Brenner) we can easily determine the law of contraction, which holds equally for the normal motor nerves and for the muscles.

With a very weak current no noticeable excitement takes place. If we gradually increase the strength of the current, the first weak contraction of the muscle occurs at the closure of the cathode—that is, when the current is closed so that the cathode is made the testing pole. On opening the cathode, or on closing or opening the anode, nothing follows. If we increase the strength of the current still more, the cathodic closure contractions become

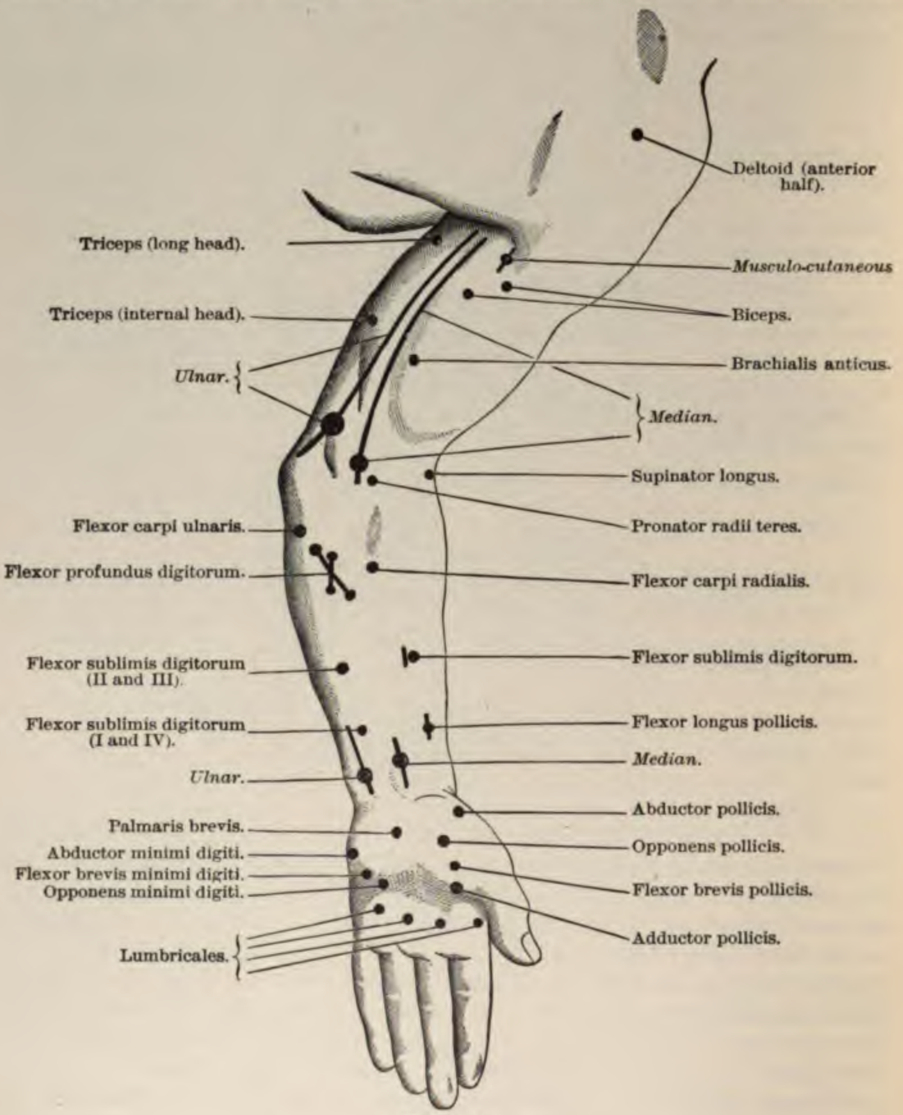

Fig. 130.

stronger, and the anodic closure and anodic opening contractions appear, now the one being earlier and stronger, and now the other. Opening of the cathode has still no effect. Only with a very strong current, in which the cathodic closure contractions have already become tetanic—that is, they still persist after the closure of the current—can we provoke weak cathodic opening contractions. Expressed in the abbreviations now in general use in electrical diagnosis, the law of contraction for normal muscles and nerves in man is as follows:[1]

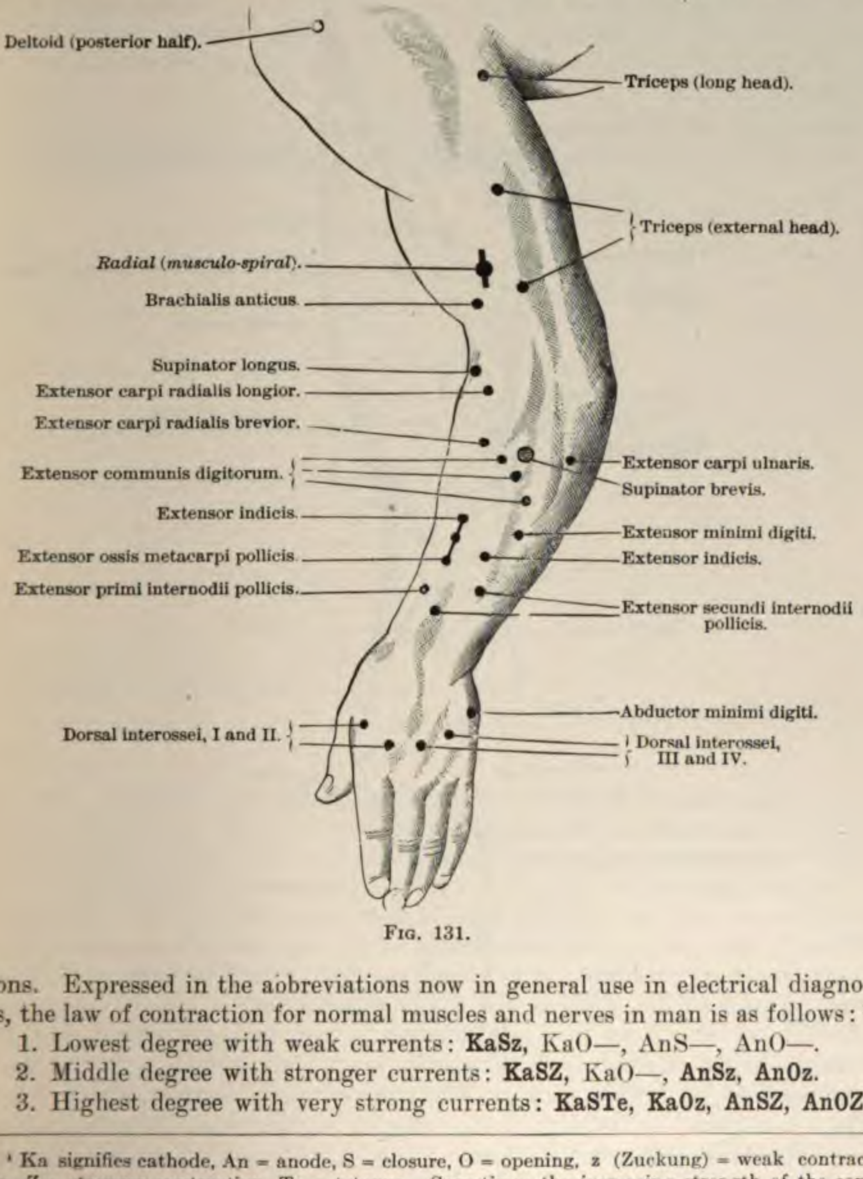
Deltoid (posterior half).
Triceps (long head).
Triceps (external head).
Radial (*musculo-spiral*).
Brachialis anticus.
Supinator longus.
Extensor carpi radialis longior.
Extensor carpi radialis brevior.
Extensor communis digitorum.
Extensor carpi ulnaris.
Supinator brevis.
Extensor indicis.
Extensor minimi digiti.
Extensor ossis metacarpi pollicis.
Extensor indicis.
Extensor primi internodii pollicis.
Extensor secundi internodii pollicis.
Abductor minimi digiti.
Dorsal interossei, I and II.
Dorsal interossei, III and IV.

FIG. 131.

1. Lowest degree with weak currents: **KaSz**, KaO—, AnS—, AnO—.
2. Middle degree with stronger currents: **KaSZ**, KaO—, **AnSz**, **AnOz.**
3. Highest degree with very strong currents: **KaSTe, KaOz, AnSZ, AnOZ.**

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[1] Ka signifies cathode, An = anode, S = closure, O = opening, z (Zuckung) = weak contraction, Z = stronger contraction, Te = tetanus. Sometimes the increasing strength of the contractions is abbreviated by the signs Z, Z' and Z''. [Many English and American writers on electricity use letters derived from the English names. Thus, C stands for cathode, closure, and contraction. A and O have the same meaning. It seems to me clearer and conducive to greater harmony to retain the German abbreviations, which are simple and definite.—K.]

The variations from the normal state seen under pathological conditions consist of quantitative and also of qualitative changes in the law of contraction. We term the simple increase or diminution of the electrical excitability in nerves or muscles, without simultaneous changes in the quality and order of the occurrence of muscular contractions, quantitative changes. The discovery of increased or diminished excitability of nerve and muscle can be made most easily in unilateral diseases, where we can compare the strengths of current required to obtain the minimal contraction on the diseased and healthy sides with each other. This is now determined without any special difficulty, since the general introduction of the absolute galvanometer. With the galvanometer thrown out of circuit we find with how many cells or at what position of the rheostat the first distinct cathodic closure contraction (KaSZ) occurs. Then the galvanometer is put into the circuit and the strength of the current in milliampères is read off directly. By extensive investigations on healthy persons Stintzing has found certain limits within which the normal excitability of the different nerves varies. Deviations from these figures above or below normal accordingly permit us to recognize an increase or a diminution of the galvanic excitability. It is to be borne in mind that Stintzing uses a small "normal electrode" with a surface of 3 sq. cm. Some of the most important averages found by Stintzing are shown in the following table:

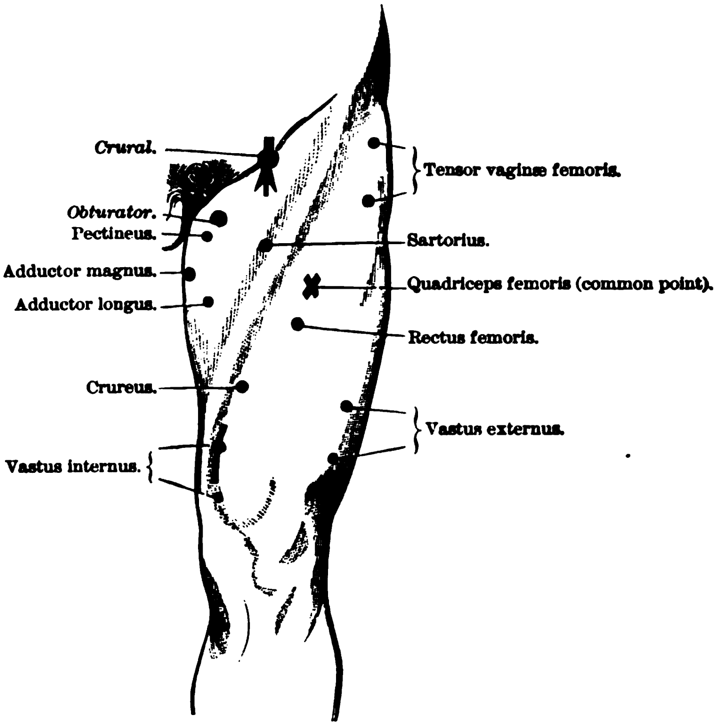

FIG. 132.

Facial nerve	1.0 to 2.5	milliampères.
Accessory nerve	0.1 to 0.4	“
Ulnar nerve	0.2 to 0.9	“
Median nerve	0.3 to 1.5	“
Radial nerve	0.9 to 2.7	“
Crural nerve	0.4 to 1.7	“
Peroneal nerve	0.2 to 2.0	“
Tibial nerve	0.4 to 2.0	“

An accurate measurement of the absolute strength of the faradic current is possible, but it has as yet been but little introduced into practice. We usually content ourselves, as we have said, with the statement of the coil distance at which the first perceptible contraction occurs. Stintzing found as an average position for the frontal nerve, 128.5 mm.; for the accessory nerve, 137 mm.; for the radial nerve, 105 mm.; for the median nerve, 122.5 mm.; for the ulnar nerve, 130 mm.; for the crural nerve, 111.5 mm.; for the

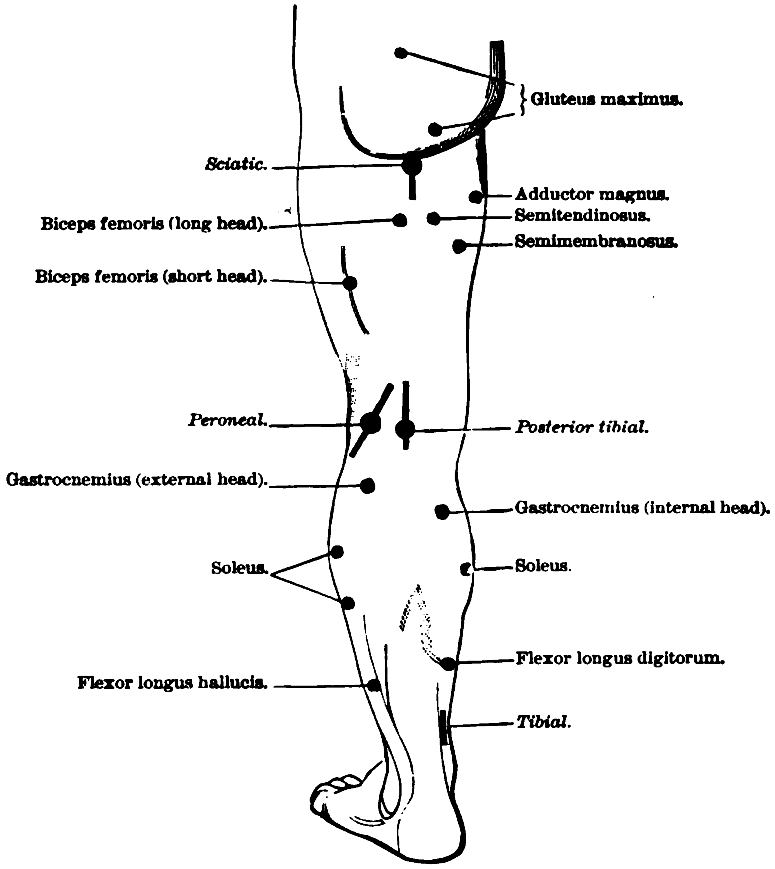

FIG. 133.

peroneal nerve, 115 mm.; and for the tibial nerve, 107.5 mm. We must refer to the special works for further details.

Changes in the quantitative electrical excitability are not uncommon in pathological conditions, but they have no very great practical diagnostic significance. A pathological increase of electrical excitability is seen especially in tetany, a diminution in many cases of neuritis, and sometimes also in myelitis, pressure paralyses, etc. The apparent diminution of excitability is often due only to increased resistance (dry, thick skin, thick layer of fat, etc.).

Much more important, however, than the simple quantitative changes of electrical excitability are those not merely quantitative but also qualitative deviations from the normal law of contraction, which were first discovered in certain forms of paralysis by Baierlacher in 1859, and were soon generally confirmed. Erb was the first to study them more accurately, and has given them the name of the "reaction of degeneration," because they are closely connected with the progress of certain anatomical changes in the paralyzed muscles and nerves.

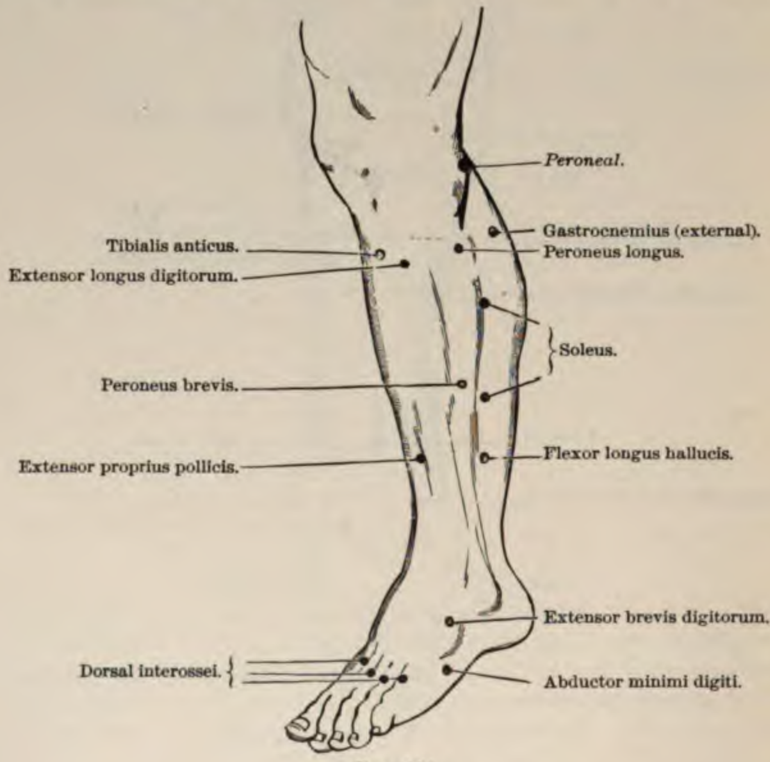

FIG. 134.

In order to make the relations of the reaction of degeneration clear, let us select as an example any fresh peripheral paralysis and follow the changes in excitability to the two currents in the nerves and muscles. In a short time (two or three days) after the onset of the paralysis a gradually increasing decline in the faradic and galvanic excitability in the nerve begins. After one or two weeks the excitability is completely lost, so that from the nerve we can no longer provoke any trace of muscular contraction with the strongest faradic or constant current. During this time the excitability of the paralyzed muscles to the faradic current has also rapidly diminished, and finally has wholly disappeared. The case is quite different with galvanic excitement of the muscles. Here we find at first a slight diminution, which in the second week passes to a decided increase of the galvanic muscular excitability. We now obtain marked muscular contractions with relatively very weak currents. Besides that, two other very important peculiarities are to be noted: 1. The muscular contractions are first of all not short and lightning-like, as under normal conditions, but they seem quite sluggish, protracted, "worm-like," and often persist during the whole duration of the closure of the current. 2. The muscular contractions occur not only chiefly at cathodic closure (KaS), as under normal conditions, but the anodic closure contractions (AnSZ) are as strong as the cathodic closure contractions (KaSZ), or even plainly exceed them. With stronger currents anodic closure tetanus (AnSTe) is readily obtained. The cathodic opening contraction (KaOZ) is also frequently stronger. 3. It may finally be mentioned here that the mechanical irritability of the muscles in such cases is usually increased, and the muscles when struck also show a slow, worm-like contraction.

This second degree of the reaction of degeneration lasts from four to eight weeks. If the paralysis be severe and long continued, or incurable, at the end of this period comes a decline of the galvanic muscular excitability. The contractions become weaker, the strength of current necessary to produce them greater, and, finally, in incurable cases, even with the strongest currents, we can obtain only a little slow anodic closure contraction (AnSZ), or none at all. It is different, however, in the milder, curable cases. In these the passage to the normal condition gradually follows either the increase of the galvanic muscular excitability, or, in more protracted cases, its secondary decline. The contractions become more vigorous and shorter, the cathodic closure contraction (KaSZ) again predominates, the faradic musculiar excitability and the faradic and galvanic excitability of the nerves finally return, and with them the old normal conditions are restored. A fact to be observed in these cases is of great interest—namely, that the voluntary motion in such cases often returns decidedly earlier than the electrical excitability of the peripheral nerves. We see, then, that a diseased nerve may be capable of conducting excitations coming from the brain, while the taking up of excitation, its direct excitability, is still completely lost. In such cases we can obtain a muscular contraction by electrical excitation of the nerve above the point of lesion.

Besides the complete reaction of degeneration just described, there is also a so-called partial reaction of degeneration, "intermediate form of reaction of degeneration," which is not infrequent in milder cases. This may show itself in several forms, but it is chiefly when the diminution of the faradic and galvanic excitability in the nerves and the diminution of faradic excitability in the muscles is only of a slight degree, while the characteristic changes in the direct galvanic excitement of the muscles—ready appearance of anodic closure contractions (AnSZ), and especially the noticeably slow character of the ensuing muscular contractions—are fully developed. In general the slow muscular contraction on direct galvanic irritation of the muscles is practically the most important symptom of reaction of degeneration. We sometimes see the occurrence of slow contractions on faradic excitement of nerves and muscles ("faradic reaction of degeneration"), and in some cases we even see slow muscular contractions on indirect galvanic irritation of the nerve (partial reaction of degeneration with indirect slowness of contraction). In the course of atrophic paralyses we often see that the different varieties of reaction of degeneration pass into one another as the process advances or improves (Stintzing).

Anatomical Changes of the Nerves and Muscles in the Reaction of Degeneration; its Significance in Diagnosis and Prognosis.—The great diagnostic importance of the electrical reaction of degeneration rests on the fact that it indicates to us directly the existence of an anatomical degeneration in the peripheral motor nerves. Only when the peripheral motor nerve has actually become degenerated does it lose its electrical (galvanic and faradic) excitability, and the corresponding muscle, all the nerves of which have become degenerated, can no longer be made to contract with the faradic, but only with the galvanic, current. This latter fact in all probability depends upon the circumstance that faradic "muscle stimulation," after all, is produced only by stimulation of the intramuscular nerve branches. A muscle deprived entirely of its nerves cannot be excited by the faradic current. The muscular tissue itself reacts only to the galvanic current, and exclusive galvanic muscle stimulation is characterized by slow contractions and the preponderance of anodic closure contractions (AnSZ), differing in these particulars from the galvanic stimulation of the normal muscle not deprived of its nerves, in which the stimulation always affects the intramuscular nerve branches as well.

It follows from this that the electrical reaction of degeneration can occur only in paralyses which are produced by a disease of the peripheral motor nerve, i. e., in the so-called "atrophic palsies" (*vide supra*). The primary site of the degeneration may be located, then, either in the peripheral prolongation of the spinal-motor ganglion cell ("peripheral paralysis"), or in the spinal-motor ganglion cells of the anterior horn itself ("atrophic spinal paralysis").

If we have to do with a peripheral paralysis, such as a traumatic lesion of a nerve trunk, the portion of the nerve peripheral to the point of lesion is separated from its cell of origin in the cord, and begins to undergo secondary degeneration. The first anatomical sign of the degeneration is a breaking down of the medullary sheath, at first into large and gradually into smaller flakes and drops. The axis cylinder is also soon destroyed, so that the sheath of Schwann finally incloses only approximately homogeneous fluid contents, which are in great part rapidly absorbed. At the same time there is an increase of the nuclei in the sheath of Schwann, and this increase, when the process is long continued, leads to a decided increase of the interstitial connective tissue in the nerve. We can readily understand that such a completely destroyed nerve can be excited neither by the faradic nor by the galvanic current.

In the muscle also, the nerve of which is degenerated, marked atrophic changes soon appear, which, contrary to the nerve degeneration, are readily apparent to the naked eye, and have given rise therefore to the designation of "atrophic paralysis." The cause of the secondary atrophy of the muscle was formerly looked for in the loss of a particular "trophic" influence which the peripheral motor neuron was believed to exercise over the muscle with which it was closely associated. However, it is not at all unlikely that this "trophic influence" is wholly identical with the functional stimulation of the muscle by the nerve, and that, therefore, the complete functional inactivity of the muscle (after the abolition of all reflex influences and the loss of muscle tone) is the real cause of the rapidly appearing muscular atrophy. Histologically the atrophy of the muscle appears primarily as a narrowing of the individual muscular fibrillæ. The cross striations may be preserved for a long time, or even altogether, and only in atrophies of very long standing does a granular or fatty degeneration of the muscle fibers finally develop. Some muscular fibers show a peculiar yellow homogeneous consistency which is called "waxy degeneration." Fatty or marked granular degeneration of the fibers occurs only in the later stages. The motor end-plates of the nerves in the muscles are spared for a comparatively long time, and they disappear only when the degeneration of the muscles has reached the highest degree.

In incurable paralyses the processes of degeneration just described gradually advance, but, in the cases that recover, a number of processes of regeneration begin sooner or later. We cannot here go into the finer details. We would mention merely that "regeneration" in all probability takes place principally only from the central nerve stump; that it consists, therefore, essentially of a new outgrowth from the maimed neurons. The old nerve sheaths of the cut-off or degenerated peripheral portion serve as conduction tubes. The axis cylinders grow out first, and then new medullary sheaths are formed. To what extent, if at all, that portion of the peripheral nerve which is separated from the ganglion cell takes part in the regeneration is not as yet definitely known. The end-plates of the nerves are restored very early, according to Gessler, even before the nerve fibers. The muscular fibers also increase in volume at the same time.

Voluntary motion in the previously paralyzed muscle returns hand in hand with the process of regeneration. It appears, as has been already mentioned, sooner than the normal electrical excitability of the nerves and muscles, because it can take place as soon as the axis cylinders are again formed, while the normal electrical excitability of nerves and muscles can take place only after the restoration of the medullary sheaths. We usually express this fact by saying that the reception of peripheral irritations depends upon the persistence of the medullary sheath. The destroyed medullary sheaths perhaps form a sort of isolating layer about the axis cylinders, which is only a poor conductor of the electrical current. This is the reason why in diseases of the peripheral nerves we sometimes find reaction of degeneration in muscles and nerves which have lost none of their voluntary motility. In such cases there is probably disease of the medullary sheaths without much degeneration of the axis cylinders.

The same anatomical changes which we have just described as a secondary degeneration in lesions of the peripheral motor nerves also develop if the primary disease has its seat in the anterior cornua of the gray matter of the spinal cord—that is, in the trophic centers themselves. In these cases, of course, the form of the disease has nothing to do with it. Both in the different forms of inflammation and of primary atrophy, and also in new growths, which affect the anterior gray matter of the cord, a secondary degeneration, with pronounced reaction of degeneration, develops from the anterior roots of the affected portion of the cord to the ends of the peripheral nerves, and even to the corresponding muscles. We shall also learn to recognize a number of primary degenerations of the peripheral nerves, such as primary neuritis, diphtheritic and toxic paralyses, etc. In such cases we find almost precisely the same anatomical changes as in secondary degenerations, and, in consequence of these changes, there is also electrical reaction of degeneration in the paralyzed nerves and muscles. In all cerebral paralyses, however, and in those spinal paralyses where the cause of the paralysis is situated above the part of the anterior gray cornua concerned, the degeneration of the peripheral nerves, and also the reaction of degeneration, are entirely wanting.

In regard to prognosis, the reaction of degeneration teaches us that anatomical changes have taken place in the nerves and muscles, from which, under some circumstances (that is, if the ganglion-cell bodies are still preserved), a restoration is still very possible, but at all events it can take place only after the lapse of a considerable time, at least two or three months. We shall soon learn to recognize a number of mild peripheral paralyses in which there is generally no reaction of degeneration. From the absence of reaction of degeneration we can then draw the conclusion, with certainty, that no coarse anatomical changes are present in the nerve, and that we may expect a much more rapid recovery from the trouble, perhaps in three or four weeks. The partial reaction of degeneration, above mentioned, is also an important symptom in regard to prognosis. It shows probably that slight changes have occurred only in the terminal branches of the muscle nerves, while the larger nerve trunks have remained approximately normal. The latter have therefore retained their electrical excitability, while the terminal branches of the nerves have lost it. The partial reaction of degeneration, therefore, always permits a more favorable prognosis in regard to time than the complete reaction of degeneration.

6. TABLE OF THE DIFFERENT FORMS OF VOLUNTARY MOTION AND THE MUSCLES AND NERVES CONCERNED THEREIN

In order to obtain an accurate knowledge of the extent of the paralysis in complicated paralytic conditions (hemiplegia, paraplegia, muscular atrophy, etc.), it is imperatively necessary to test all the muscle groups in order. The performance of these tests and their diagnostic value and significance will be materially aided by the use of the following table, arranged according to movements.

We must, however, observe that our knowledge concerning the nuclear origin of the different spinal nerves, in many instances, is still uncertain, and that, moreover, the different nuclei are generally distributed over several segments of the cord and merge into one another.

I. MUSCLES OF THE HEAD AND TRUNK

1. *Facial Muscles and Muscles of Mastication*

1. Wrinkling the forehead transversely (Mm. frontalis et occipitalis, N. facialis).
2. Wrinkling the forehead longitudinally (M. corrugator supercilii, N. facialis).
3. Closing the eyes (M. orbicularis palpebrarum, N. facialis).
4. Dilating the nostrils (M. compressor nasi et M. levator alæ nasi, N. facialis).
5. Drawing the mouth outward and upward (Mm. levator labii superioris, zygomatici, risorius, N. facialis).
6. Drawing the mouth downward (M. depressor anguli oris et labii superioris, N. facialis).
7. Raising the lower lip (M. levator menti, N. facialis).
8. Protruding the lips and whistling (M. orbicularis oris, N. facialis).
9. Chewing (Mm. masseter et temporalis, motor branch of N. trigeminus).
10. Lateral and forward movements of the lower jaw (Mm. pterygoidei externi et interni, N. trigeminus).

2. *Ocular Muscles*

1. Raising the upper lid (M. levator palpebræ superioris, N. oculomotorius).
2. Looking upward and inward (M. rectus superior) and upward and outward (M. obliquus inferior, N. oculomotorius).
3. Looking inward (M. rectus internus, M. oculomotorius).
4. Looking outward (M. rectus externus, N. abducens).
5. Looking downward and inward (M. rectus inferior, N. oculomotorius).
6. Looking downward and outward (M. obliquus superior, N. trochlearis).
7. Contraction of the pupil (M. sphincter iridis, N. oculomotorius).
8. Accommodation (M. ciliaris, N. oculomotorius).

3. *Tongue. Soft Palate. Pharynx. Larynx*

1. Tongue movements (N. hypoglossus).
2. Soft palate (N. vago-accessorius).
3. Swallowing (N. vago-accessorius).
4. Epiglottis (Mm. thyreo-epiglotticus et ary-epiglotticus, N. laryngeus superior).
5. Vocal cords (N. recurrens).

4. *Head and Trunk*

1. Flexion of the head and cervical vertebræ forward (Mm. recti capitis antici, longus colli rectus, N. cervicales 1 to 3), M. sterno-cleido-mastoideus (N. accessorius).
2. Movement of the head and cervical vertebræ backward (Mm. splenius

capitis et colli, biventer complexus, recti capitis postici, spinalis et semispinalis cervicis, N. cervicales 1 to 4).

3. Turning the head (M. sterno-cleido-mastoideus, N. accessorius, M. obliquus capitis inferior et obliquus colli).

4. Lateral flexion of the head (Mm. recti capitis laterales, M. spinalis cervicis).

5. Extension of the vertebral column (Mm. sacro-lumbalis et longissimus dorsi, M. spinalis dorsi).

6. Flexion of the vertebral column forward and raising the trunk from a recumbent position (abdominal muscles). Recti abdominis from 8 D. N.; also obliqui abdominis.

7. Torsion of the vertebral column (M. semispinalis dorsi).

8. Lateral flexion of the vertebral column (M. quadratus lumborum, innervated from the crural plexus, Mm. intertransversarii).

9. Diaphragm (N. phrenicus from N. cervicales 4 and possibly also 5).

II. SHOULDER BLADE AND UPPER EXTREMITY

1. *Movements of the Shoulder Blade*

1. Raising the shoulder blade (M. trapezius, N. accessorius, M. levator anguli scapulæ, 1 to 3 C. N.[1]).

2. Drawing the shoulder blade toward the median line (Mm. rhomboidei, Nn. thoracici posteriores from 4 and 5 C. N.).

3. Fixation and rotation of the shoulder blade in raising the upper arm to a vertical position (M. serratus anticus major, N. thoracicus major, principally from 5 C. N.).

2. *Movements at the Shoulder*

1. Raising the arm forward and outward (M. deltoideus, N. axillaris[2] [circumflex] from 5 and 6 C. N.).

2. Adduction and drawing downward (Mm. pectorales major et minor, N. thoracici anteriores from 5 and 6 C. N., M. latissimus dorsi, N. subscapularis longus from 5 and 6 C. N.).

3. Outward rotation (Mm. supraspinalis, infraspinalis, teres minor, N. suprascapularis from 4 and 5 C. N.).

4. Inward rotation (Mm. subscapularis et teres major, N. subscapularis from 5 and 6 C. N.).

3. *Movements at the Elbow*

1. Flexion (Mm. biceps et brachialis anticus, N. musculo-cutaneous; 5 and 6 C. N., M. supinator longus, N. radialis [musculo-spiralis]; 6 and 7 C. N.).

2. Extension (M. triceps, N. radialis, 6 and 7 C. N.).

3. Supination (M. biceps, N. musculo-cutaneous, M. supinator brevis, N. radialis; 5 C. N.).

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[1] C. N. = cervical nerve, L. N. = lumbar nerve, S. N. = sacral nerve, that is, the several anterior roots arising from the corresponding segment of the spinal cord.

[2] [We have followed Henle's nomenclature for the peripheral nerves.—K.]

4. Pronation (Mm. pronator teres et pronator quadratus, N. medianus; 6 C. N.).

4. *Movements at the Wrist*

1. Dorsal extension (Mm. extensor carpi radialis longus et brevis, extensor carpi ulnaris, N. radialis; 6 and 7 C. N.).

2. Volar flexion (Flexor carpi radialis, N. medianus. Flexor carpi ulnaris, N. ulnaris; 7 and 8 C. N.).

3. Radial flexion (M. extensor carpi radialis longus).

4. Ulnar flexion (Mm. flexor et extensor carpi ulnaris).

5. *Movements of the Second and Third Fingers*

1. Extension of the proximal phalanges (Mm. extensor digitorum communis, extensor indicis, extensor minimi digiti, N. radialis; 6 and particularly 7 C. N.).

2. Extension of the distal phalanges [phalangines and phalangettes] (M. interossei, N. ulnaris).

3. Flexion of the proximal phalanges (Mm. interossei et lumbricales, Nn. ulnaris et medianus; 7 and particularly 8 C. N.).

4. Flexion of the distal phalanges (Mm. flexor digitorum sublimis et profundus, the latter for the third phalanx [phalangette], N. medianus; 7 and particularly 8 C. N.).

5. Spreading the fingers, abduction (Mm. interossei externi [dorsales], N. ulnaris; 8 C. N.).

6. Adduction of the fingers (Mm. interossei interni [palmares], N. ulnaris).

7. Flexion of the proximal phalanx and abduction of the little finger (Mm. flexor brevis et abductor digiti minimi in the hypothenar eminence, N. ulnaris).

6. *Movements of the Thumb*

1. Extension of the metacarpal and both phalanges (Mm. extensor pollicis brevis et longus, the latter for the second phalanx, N. radialis; 8 C. N.).

2. Abduction of the metacarpus (M. abductor pollicis longus, N. radialis).

3. Adduction of the metacarpus (M. adductor et caput profundum flexoris brevis, N. ulnaris).

4. Flexion and opposition of the metacarpus, flexion of the first phalanx with simultaneous extension of the distal phalanx (Thenar muscles: Mm. opponens, abductor brevis et caput superficiale flexoris brevis, N. medianus; 8 C. N.).

5. Flexion of the second phalanx (M. flexor pollicis longus, N. medianus).

7. *Movements of the Little Finger*

1. Flexion and abduction of the proximal phalanx (muscles of the hypothenar eminence; 8 C. N. and particularly 1 D. N.).

III. LOWER EXTREMITY

1. *Movements at the Hip*

1. Flexion (Mm. ilio-psoas, sartorius, N. cruralis, M. tensor vaginæ femoris, N. glutæus superior; 1 and 2 L. N.).
2. Extension (M. glutæus maximus, N. glutæus inferior from the sciatic plexus; 1 S. N.).
3. Abduction (M. glutæus medius et minimus, N. glutæus superior; 1 S. N.).
4. Adduction (Mm. adductor brevis, adductor longus, adductor magnus, pectineus, gracilis, N. obturatorius from lumbar plexus; 2 and 3 L. N.).
5. Outward rotation (Mm. pyriformis, obturator internus, gemelli, quadratus femoris, N. ischiadicus, M. obturator externus, N. obturatorius from lumbar plexus; 5 L. N.).
6. Inward rotation (Mm. glutæus medius et minimus, N. glutæus superior; 1 S. N.).

2. *Movements at the Knee*

1. Extension (M. extensor cruris quadriceps, N. cruralis; 3 and particularly 4 L. N.).
2. Flexion (Mm. biceps, semimembranosus et semitendinosus, N. ischiadicus; 5 L. N. and 1 S. N.).

3. *Movements at the Ankle and of the Toes*

1. Dorsal extension of the inner edge of the foot (M. tibialis anticus, N. peroneus; 4 and 5 L. N.).
2. Dorsal extension of the outer edge of the foot (Mm. peroneus longus et brevis, N. peroneus; 5 L. N. and 1 S. N.).
3. Plantar flexion of the foot (Mm. gastrocnemius et soleus, N. tibialis; 1 and 2 S. N.).
4. Adduction of the inner edge of the foot (M. tibialis posticus, N. tibialis).
5. Abduction of the foot (M. peroneus brevis, N. peroneus).
6. Dorsal extension of the toes (Mm. extensor digitorum communis et extensor hallucis longus, N. peroneus; 4 and 5 L. N.).
7. Plantar flexion of the toes (Mm. flexores digitorum et hallucis longi et flexor digitorum brevis, N. tibialis; 1 and 2 S. N.).

CHAPTER II

THE DIFFERENT FORMS OF PERIPHERAL PARALYSIS

1. PARALYSIS OF THE OCULAR MUSCLES

Ætiology.—The largest part of all the ocular paralyses arise from affections which involve either the peripheral oculomotor nerves or their nuclei in the brain stem. We accordingly make a distinction between peripheral and nuclear paralyses of the ocular muscles. As we shall take up the latter more fully in the description of chronic bulbar paralysis, we have here to mention only the most important and most frequent causes of the peripheral ocular paralyses. These are as follows:

1. Injuries which directly affect the nerve trunks or their branches: blows on the eye, knife stabs, fractures of the skull involving the orbit or the base of the skull, and the like.

2. Compression of the nerves from morbid processes in their neighborhood. Tumors of the base of the skull, especially, very often lead to ocular paralyses. Periostitis at the base of the skull or in the orbit may also cause similar symptoms, and so may syphilitic diseases of the nerves and their surroundings, the meninges or periosteum, aneurisms of the basilar artery, acute or chronic meningitis in its different forms, etc. In all these cases we usually have to do with a purely mechanical compression of the affected nerves by the morbid new growths in their immediate vicinity. More rarely the pathological process directly invades the nerves themselves.

3. The so-called rheumatic ocular paralyses are quite common. These arise after some decided exposure to cold, such as a draught from an open window, and depend, as is supposed, upon an acute neuritis of the affected nerve, and hence correspond completely to the other rheumatic paralyses, such as rheumatic facial paralysis. Among the "rheumatic paralyses" we usually class the paralyses which apparently come on spontaneously and completely recover, for which no other special cause can be made out.

4. The ocular paralyses that sometimes arise after certain acute diseases are also of a peripheral nature, and are due to degenerative neuritis of the affected nerves. They are most frequent as a result of diphtheria, and are very much more rare in typhoid fever, acute rheumatism, etc. Of chronic diseases, diabetes mellitus may sometimes give rise to ocular paralyses, especially to paralysis of accommodation. The ocular paralyses occurring as a symptom of alcoholic polyneuritis or polioencephalitis may be referred to a toxic influence that is recognized.

5. Finally, we often see ocular paralyses as a symptom of more extensive nervous disease, especially in tabes, multiple sclerosis, etc. The details of these conditions will be found in the description of the several diseases.

Symptoms.—Since we must refer to the text-books of ophthalmology in regard to the more precise symptomatology and the more special methods of ophthalmic investigation, we will here give only a brief review of the chief symptoms of ocular paralyses which are important in nervous pathology.

The disturbance in the mobility of one eye is usually first noticed by the patient himself by the appearance of double images—double vision, or diplopia. These arise because, on looking to one side, the eye on the paralyzed side cannot be brought into the corresponding position (strabismus), and consequently the retinal images no longer fall upon the same spots, the two images are no longer projected outward in a corresponding fashion, so that two different images are therefore produced of the same external object. In pathological convergence of the visual axes homologous double images arise, in pathological divergence crossed images—that is, in the first case, on closing one eye, the image disappears on the same side, in the second case it disappears on the opposite side. By alternately fixing the gaze on one or the other of two fingers held in line with each other, and by regarding the disappearance, on closing one eye, of the double image of the finger not fixed, we can easily demonstrate this on ourselves. If, then, crossed double images arise, for example, on looking to the right, we must have a divergent strabismus—that is, an imperfect function of the left internus; but if there are homonymous double images, there must be a convergent strabismus, and consequently a weakness of the right abducens. It makes it much easier to test the double images if we put a colored glass before one of the patient's eyes. False projections of the visual field arise, in connection with the double images and with the abnormal strength of innervation which the patient has to exert for his ocular muscles, so that the patient's judgment of the position of external objects is uncertain. In the more extensive ocular paralyses this often leads to a pronounced feeling of dizziness. In order to avoid this unpleasantness many patients confine themselves to monocular vision, close the affected eye, or put their heads in a position to avoid the double images.

Finally, we must mention a symptom to be observed in almost every ocular paralysis—the so-called secondary deviation of the healthy eye. If, after the sound eye has been covered, we have the paretic eye fix itself upon a point which it cannot reach at all, or which it can reach only after the utmost exertion, we see, when the covering hand is taken away from the sound eye, that the latter has been moved much too far in the corresponding direction. The abnormal exertions of innervation with the affected eye, somewhat after the analogy of certain associated movements, pass over to the associated muscle of the healthy side and cause in it too extensive a contraction.

Physical examination gives the following results, according to the extent of the paralysis:

On complete paralysis of one *oculomotor nerve* (the levator palpebræ superioris, the superior, inferior, and internal recti, the inferior oblique, the sphincter of the iris, and the ciliary muscle) the first thing that is noticed, besides the disturbance in the movements of the eye, is the more or less complete drooping of the upper lid—ptosis. If the patient wishes to lift the upper lid he cannot do so at all or only to a slight degree. If he tries very hard there is usually an evident contraction of the corresponding frontalis muscle. If we ask the patient to follow with his eyes the motion of any object, such as the finger, held before him, while he keeps his head still, we notice at once that the affected eye does not move upward, downward, or inward. The pupil is dilated (mydriasis) and no longer contracts to light. Accommodation is lost and distinct vision for near objects is impossible. As a rule, the whole eye seems rather prominent (paralytic exophthalmus), because the backward traction of the recti is very largely absent. In old oculomotor paralysis there is often a secondary contracture in the unparalyzed external rectus (and superior oblique), by which the eye is persistently drawn outward. Partial oculomotor paralyses are not infrequent, especially isolated ptosis, also paralysis limited to the levator palpebræ and superior rectus, paralysis of the internal, inferior, or superior rectus, or, finally, isolated paralysis of accommodation, and they may usually be easily recognized from what has been said.

Paralysis of the *abducens* is characterized by the inability to move the external rectus. The eye can no longer be moved, or it can be moved only imperfectly, outward beyond the median line. In old paralysis the eye is drawn inward from a secondary contracture of the internal rectus, and convergent strabismus arises. Paralysis of the abducens may often be isolated, but it is sometimes bilateral, or combined with other ocular paralyses.

Paralysis of the *trochlear nerve,* the superior oblique muscle, is not perfectly easy to recognize, but it is rarely of special practical importance. The action of the superior oblique coincides mainly with that of the inferior rectus. If the trochlear be paralyzed the eye turns somewhat inward on looking downward, because the superior oblique acts also to abduct the eyeball, and therefore acts chiefly on looking downward and outward. The paralysis of the trochlear nerve is sometimes made manifest by the failure of the eye to revolve, which rotation normally takes place on looking downward, and is due to the superior oblique muscle. This latter movement takes place in each eye about a sagittal axis, in such a way that the left eye is turned from the left and upward to the left and downward, and the right eye from the right and upward to the right and downward. In regard to diagnosis, it is also characteristic that the double images in trochlear paralysis appear only in the lower half of the field of vision, and especially on looking downward. Hence it happens that the disturbance of vision is especially manifest in going up or down stairs, because in such cases the double images of the steps are disturbing.

The following must be added in regard to the separate clinical forms of ocular paralysis: Rheumatic ("neuritic") ocular paralysis affects the abducens most frequently, and not infrequently the oculomotor or one of its branches, as in isolated ptosis. We have seen a rare case in which exposure to severe cold had been followed by a complete paralysis of all the external muscles of the right eye, with complete ptosis, and almost absolute immobility of the eye in all directions. The rheumatic ocular paralyses are almost always acute in their onset, and they are often during the first period associated with sensations of pain about the eye and in the head. Vomiting (of reflex origin?) is also not rare at the beginning of the affection. The course of most cases is favorable, since they usually completely recover in a few weeks, though sometimes not for months. In some cases the paralysis may remain stationary.

The diphtheritic ocular paralyses usually appear, like the other diphtheritic paralyses, a week or two after the termination of the disease. They most frequently affect the muscles of accommodation, so that the patient complains chiefly of indistinct vision for near objects; but we sometimes see paralyses of the external ocular muscles also, the abducens or the internal rectus. The prognosis of diphtheritic paralyses is almost invariably favorable.

Finally, we must mention here the so-called "*recurrent or periodical oculomotor paralysis,*" to which Möbius and others have lately called attention, and whose nature is still almost wholly inexplicable. In the cases of this sort repeated paralyses of one oculomotor nerve, often associated with headache and vomiting, just as in migraine, come on at longer or shorter intervals (in women sometimes at the menstrual period) in the same individual, who has often had them since childhood. All branches of the oculomotor are usually affected alike, but sometimes the internal ocular muscles are spared. In milder cases there may be only ptosis. The individual attack sometimes lasts only a few days, but often some weeks. The attacks usually become gradually more severe later on. The nature of recurrent oculomotor paralysis is still quite unknown, but it seems to be growing more probable that in a part of the cases at least the disease is closely related to migraine (*q. v.*), and forms in a measure only an unusual development of migraine—*migraine ophthalmoplégique.* The genuine cases of the disease, however, must, of course, be distinguished from the recurrent oculomotor paralysis that sometimes occurs in tabes, cerebral syphilis, tumors of the base of the skull, etc. [The relation to migraine seems very doubtful. There is rarely complete recovery between the attacks, and there is a tendency for the attacks to increase in frequency and duration as well as in severity. In three cases which have come to autopsy lesions have been found affecting the nerve—tumors or inflammatory thickening. Some of the cases have also had anæsthesia in one or more branches of the distribution of the trigeminus during the attack. Besides the recurrent oculomotor paralysis and recurrent facial paralysis (*vide infra*), recurrent trochlear, and abducens paralyses and recurrent paralysis of several cranial nerves at once have been reported.—K.]

Nothing generally applicable can be said as to the course and prognosis of the other forms of ocular paralyses, since in them everything depends upon the form of the underlying disease.

Treatment.—In regard to the fulfillment of any possible causal indication we must remember especially that ocular paralysis is not very infrequently of syphilitic origin. Iodid of potassium and energetic mercurial inunction may sometimes give very good results in such cases. Hence these remedies must also be tried in doubtful cases.

Of other remedies galvanic treatment gives the speediest relief. We pass weak currents transversely through the temples, or, what is usually better, we put the anode to the back of the neck and apply the labile cathode to the closed eye, especially to the region corresponding to the paralyzed muscles. Great caution, weak currents, and the avoidance of any great variations in the currents are, of course, necessary. We may also try preparations of strychnin internally or subcutaneously. We must refer to special treatises with regard to a correction of the double images by covering the affected eye, spectacles with one dark or ground glass, or prismatic spectacles, or in regard to operations, such as tenotomy, that are sometimes performed.

2. PARALYSIS OF THE MOTOR BRANCH OF THE TRIGEMINUS.

(Paralysis of the Muscles of Mastication)

Paralysis of the muscles of mastication, the masseters and temporals, supplied by the third branch of the trigeminus, is a rare affection. It is most frequently seen in diseases of the base of the skull which compress the motor branch of the fifth. We shall also learn later on to recognize paralysis of the muscles of mastication as a rare symptom of chronic bulbar affections.

The chief symptom of motor paralysis of the trigeminus is the difficulty or impossibility of chewing. In unilateral paralysis the patient can chew only on the healthy side; in bilateral paralysis he can no longer chew at all. Paresis of the muscles of mastication can be readily determined by the lack of strength in resisting the attempt to draw down the lower jaw or by the patient's inability to make a deep imprint in biting a bit of wood (lead pencil, etc.). In complete paralysis of the muscles of mastication the lower jaw hangs loosely down, and it can also no longer be moved sideways, from the coexisting paralysis of the pterygoids. We cannot usually detect clinically disturbances in the other muscles supplied by the trigeminus (mylohyoid, buccinator, tensor palati, and tensor tympani), but there are often sensory disturbances in the distribution of the trigeminus at the same time.

The prognosis and treatment depend upon the primary disease. Local faradization or galvanization of the paralyzed muscles is to be tried.

3. FACIAL PARALYSIS

(Mimetic Facial Paralysis. Bell's Palsy)

Ætiology.—Facial paralysis is one of the commonest peripheral paralyses, as we can understand from the superficial position of the nerve, exposing it to external injuries, and from its course through the narrow Fallopian canal. We consider it improbable that a special "neuropathic family predisposition" plays a part in the occurrence of peripheral facial paralysis, as Charcot's school maintains. The most important causes of facial paralysis are: (1) Exposure to cold, draughts, sleeping by an open window, riding in the cars with the window open, etc. The paralyses arising in this way are termed "rheumatic," and we also include in this class those peripheral paralyses which are apparently spontaneous—that is, those without any marked exposure to cold that can be discovered. In all these cases we probably have to do with a neuritis of the nerve trunk, which comes on in a way as yet unknown (infectious? toxic?). [In a recent autopsy of a case of so-called "rheumatic" facial paralysis, Minkowski found a simple degenerative parenchymatous neuritis, without any swelling of the nerve sheath or increase of connective tissue.—K.] (2) Diseases of the middle ear, and especially caries [and fracture] of the petrous bone. As the facial passes through the Fallopian canal, which is in the immediate vicinity of the tympanic cavity, we can easily understand how so often in caries of the petrous bone, and often perhaps in purulent affections of the middle ear, inflammation may invade the trunk of the facial, or how the facial may be compressed by inflammatory exudations, etc. (3) In rare cases a pressure paralysis of the facial nerve arises in swelling of the parotid gland or tumors in its vicinity. (4) Diseases of the base of the skull or brain, tumors, syphilitic new growths, and acute or chronic inflammations, often give rise to the development of a facial paralysis by invasion of the trunk of the facial or compression of it. Of particular interest are the tumors arising from the sheath of the acoustic nerve, which, in addition to disturbances of hearing, also very soon produce a paralysis of the facial nerve which lies in close proximity to it. (5) We shall have to speak repeatedly in the following sections of the frequent implication of the facial nerve in diseases of the brain and medulla and of the implication of the facial muscles in multiple neuritis, in juvenile muscular dystrophy, and other diseases.

Symptoms and Course.—The manifold character and different functions of the nerves which unite in the trunk of the facial are the causes of the quite large array of symptoms in facial paralysis. Most of these symptoms are manifest only in peripheral facial paralysis, and the following description, therefore, refers chiefly to this.

The paralysis of the facial muscles of expression is the most striking and characteristic (see Fig. 135). The paralyzed half of the face is lax and expressionless, the wrinkles in the forehead are obliterated, the eye is abnormally wide open, as the weight of the lower eyelid draws it down, and the eye also waters (epiphora), the nasolabial fold is obliterated, the corner of the mouth droops, and saliva frequently flows from it. The paralysis becomes still more marked on any movement of the face, on wrinkling the forehead, turning up the nose, laughing, talking, whistling, or inflating the cheeks. The eye can be only partly closed. On attempting it, the upper lid sinks down from its weight (a weakening of the levator palpebræ superioris), the eye is turned upward (so-called Bell's sign), so that the pupil is covered, but quite a wide space is left between the eyelids (lagophthalmus). This defective closure of the lids facilitates the entrance of dust, etc., into the eye, and sometimes gives rise to conjunctivitis, or even to severer inflammation of the eye. Speech is rendered difficult and indistinct from defective movements of the lips, and mastication becomes impaired from the imperfect movement of the cheeks. The patient can no longer whistle because he can no longer pucker the lips sufficiently on the affected side. If he tries to puff out the cheeks or keep them inflated, we notice plainly the flaccidity of the cheek on the paralyzed side. On drawing down the lower lip the paralysis of the platysma myoides is sometimes very plainly seen. The tongue is often apparently protruded to one side, but this is due merely to the distorted position of the mouth. Very often, however, we see, when the mouth is open and the tongue is at rest, that the half of the tongue corresponding to the paralyzed side lies deeper than the other half, a condition which is probably connected with a paralysis of the stylohyoid and digastric muscles. [In rare instances both nerves are affected, giving rise to a double facial paralysis.—K.]

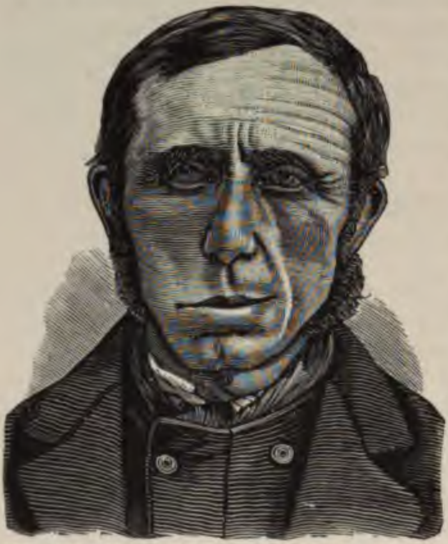

FIG. 135.—Right facial paralysis (after SEELIGMÜLLER). The folds are smoothed out and in part entirely absent on the paralyzed side, while they are strongly marked on the left. The mouth and nose are drawn to the left.

Isolated earlier reports, in which the writers claimed to have found a paresis of the soft palate (the fibers from the facial are said to pass through the superficial petrosal nerve to the spheno-palatine ganglion, and thence to the soft palate), causing the soft palate to droop more on the affected side, and to be raised obliquely toward the healthy side on phonation, are due to an error. According to more recent investigations, the soft palate is innervated only by fibers from the vagus-accessory and not from the facial.

Disturbances of taste in the anterior two thirds of the tongue have been repeatedly found on the paralyzed side, but they usually attain only a slight degree. They are explained by an affection of the fibers of the chorda tympani, which run for some distance in the facial, as has been described on page 191. At the beginning of the paralysis many patients complain of subjective sensations of taste. Later on the blunting of taste may often be discovered by careful testing. Tactile sensibility in the tongue is only exceptionally diminished (sensory fibers in the chorda?). A diminished secretion of saliva (fibers in the chorda), on the paralyzed side, is not infrequently seen; exceptionally also an increase. Disturbances of the sweat secretion on the paralyzed side occur more frequently, especially anhidrosis. If the lesion be located in the region of the geniculate ganglion, a diminution of the lachrymal secretion is always found (G. Köster). Disturbances of hearing are frequent, due either to some complicating aural trouble (*vide supra*) or to a coexisting affection of the acoustic nerve. Paralysis of the stapedius muscle, however, sometimes seems to cause symptoms, including a marked sensitiveness to all loud sounds, and even an abnormal acuteness of hearing, especially for low notes (hyperacusis, oxyokoia). These symptoms are due to the fact that in paralysis of the stapedius its antagonist, the tensor tympani, causes a greater tension of the membrana tympani. [According to Hunt the facial nerve is a mixed nerve, the nerve of Wrisberg forming its sensory portion. This sends sensory fibers to the skin just in front of the ear and to the external auditory canal. Lesions involving this portion of the nerve may give rise to anæsthesia, pain, and herpetic eruption in this area. —K.] Reflex movements, winking, etc., are, of course, lost in complete peripheral facial paralysis. The special reflexes, which are often seen in the later stages of facial paralysis, will be discussed later on.

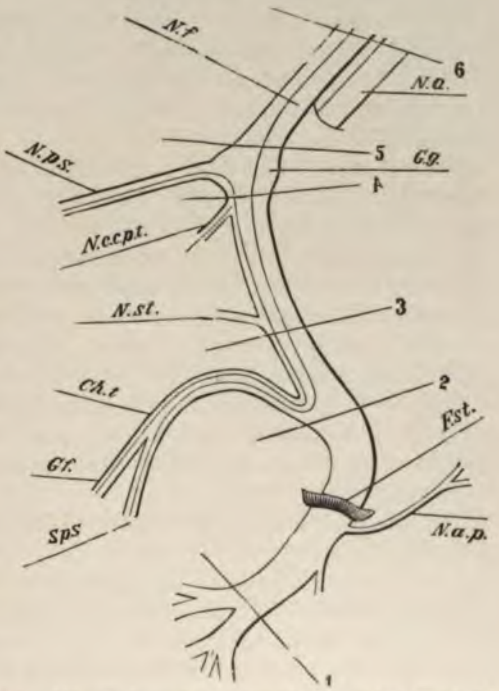

Fig. 136.—Schematic representation of the trunk of the facial from the base of the skull to the pes anserinus. Different localizations of the lesion in paralysis. *N.f.* Facial nerve. *N.p.s.* Great superficial petrosal. *N.c.c.p.t.* Nerve communicating with the tympanic plexus. *N.st.* Stapedius. *Ch.t.* Chorda tympani. *G.f.* Fibers of taste. *Sps.* Nerve governing the secretion of saliva. *N.a.* Acoustic nerve. *G.g.* Geniculate ganglion. *F.st.* Stylomastoid foramen. *N.a.p.* Posterior auricular nerve.

By testing all the symptoms described, in most cases we can decide with accuracy upon the place where the break in conduction in the facial must occur. If we examine the accompanying plan of the facial (Fig. 136), devised by Erb, we can easily understand the following chief symptomatic forms of facial paralysis. Of course we must not forget that many points (innervation of the soft palate, of the secretion of saliva and tears, etc.) are still in need of further investigation.

(1) Paralysis of the facial muscles; but taste, secretion of saliva, and hearing normal; seat of the affection in the portion between 1 and 2, usually the trunk of the facial below the Fallopian canal.

(2) Paralysis of the facial muscles, disturbance of taste, and eventually diminished secretion of saliva, but hearing normal; seat of the affection within the Fallopian canal between 2 and 3.

(3) Paralysis of the facial muscles, disturbance of taste, diminished secretion of saliva, abnormal acuteness of hearing; seat between 3 and 4.

(4) Paralysis of the facial muscles, disturbance of taste, diminished secretion of saliva and tears, abnormal acuteness of hearing, and paresis of the soft palate (*? vide supra*); seat in the geniculate ganglion between 4 and 5.

(5) Paralysis of the facial muscles, diminished secretion of saliva, abnormal acuteness of hearing, but no disturbance of taste; seat above the geniculate ganglion between 5 and 6.

The changes in electrical excitability, and some other changes, may be best described in connection with the course of facial paralysis. Ordinarily rheumatic paralysis begins quite suddenly; less frequently the onset is more gradual. Sometimes there are, for a short time, subjective prodromata, such as abnormal sensations of taste, slight ringing in the ears, and very often painful sensations in the ear, behind the ear, and in the face, which symptoms may be referred to the beginning of acute inflammatory processes in the nerve. In a few cases the occurrence of herpes vesicles has been observed in the distribution of the affected facial, a condition which may be most readily explained, in accordance with what was said on page 176, by reference to the numerous anastomoses between the branches of the facial and those of the trigeminus.

In regard to the general course of facial paralysis we distinguish the three following forms:

(1) *The mild form of facial paralysis,* to which especially many rheumatic paralyses belong. The affection is usually referred only to the facial muscles, disturbances of taste, etc., being wholly absent. Electrical excitability in the facial and the paralyzed muscles remains entirely normal. Recovery is rapid, usually in two or three weeks. In these cases we may certainly suppose that there are no severe and deep-seated anatomical changes in the nervous or muscular fibers.

(2) *The middle form of facial paralysis* (Erb). In this there is no complete reaction of degeneration, but only a partial one. The excitability of the nerve is somewhat diminished, but it is not lost. In the muscles, however, in about two or three weeks, there appears a decided increase of galvanic excitability to direct excitement. The anodic closure contraction (AnSZ) is also greater than the cathodic closure contraction (KaSZ), and the contractions are slow. In regard to prognosis we may decide from this that the recovery will still be quite rapid. It usually follows in from four to six weeks.

(3) *The severe form of facial paralysis* is that in which there is a complete reaction of degeneration in the nerve and muscles, the details of which we

have learned in the previous chapter—loss of faradic and galvanic excitability of the nerve, loss of faradic excitability of the muscles, and quantitative and qualitative changes in the galvanic excitability of the muscles. In this form there are always coarse processes of degeneration in the nerve and atrophy of the muscles, so that recovery follows, if at all, only after three to six months, or even later, because the processes of regeneration require at least as much time for their accomplishment. We often see in the later stages of these cases special symptoms of motor irritation (Hitzig). These consist, first, of a slight or sometimes very pronounced tonic contracture of the paralyzed muscles, which is sometimes very striking; if such cases are examined carelessly, we may easily mistake the (flaccid) healthy side of the face for the paralyzed side; second, of single spasmodic contractions of the muscles, a true facial tic (*vide infra*) following the paralysis of the facial; third, of special associated movements—if the patient closes his eyes, winks, etc., there always follows a marked distortion of the corner of the mouth, which cannot be suppressed; we have seen this symptom frequently in students in whom a peripheral branch of the facial has been injured in a duel; fourth, of an increased reflex irritability—on pricking the skin, or blowing on it, vigorous muscular contractions follow. We have often ourselves seen contractions in the affected facial muscles following a blow on the bridge of the nose, on the nasal bone, or on the forehead on the healthy side. These reflexes come from the skin, or perhaps in part from the periosteum and the fasciæ also. All these symptoms—the contracture, the abnormal twitchings, and the associated movements—may last for a very long time—for years in incurable or in imperfectly cured cases.

Prognosis.—The prognosis of facial paralysis depends, of course, in the first place, upon the primary disease, if any exists. Paralysis in tumors of the base of the brain, caries of the petrous bone, etc., is almost always incurable. The course of the paralysis in affections of the middle ear depends upon the curability of the latter disease. Very important data for the accurate prognosis of rheumatic paralysis are given by the electrical examination, as has been carefully described above. Of course, we can never form a definite judgment from this at the beginning of the paralysis, but only at the end of the first week. If, at the end of the first week or fortnight, the electrical excitability of the nerve still remains normal, we can almost certainly prophesy a rapid and favorable course. If reaction of degeneration appears, we cannot count upon recovery in the most favorable cases before two or three months. Relapses almost never follow directly upon the course of a paralysis, but it is by no means rare to see peripheral facial paralysis occur several times in the same patient after intervals of some years. We have seen a man of about thirty who had a peripheral facial paralysis four times within a few years, which disappeared each time after a few weeks—a condition which possibly is to be regarded as analogous to the "periodical oculomotor paralysis" (see page 229).

Diagnosis.—The symptoms of facial paralysis are so pronounced that the paralysis itself can always be easily recognized. In regard to the precise form of the paralysis and its cause, we can often decide only by considering the ætiological factors, such as injuries, exposure to cold, or aural affections. In distinguishing between peripheral and central (bulbar or cerebral) paralyses, the other coexisting bulbar or cerebral symptoms must first of all be considered. We shall learn to recognize more accurately later the different modes in which, in these cases, facial paralysis may be combined with paralysis of the other cerebral nerves or the nerves of the extremities. In doubtful cases electrical examination is often of decisive value. Reaction of degeneration can be present only in peripheral paralysis, or in such bulbar paralyses as affect the fibers of the facial below the facial nucleus or affect the nucleus itself. In all cerebral paralyses the electrical excitability is perfectly retained. It may also be mentioned here briefly that in all cerebral facial paralyses the frontal muscles and the orbicularis palpebrarum are unaffected, while in nuclear and peripheral paralyses they also are paralyzed.

Treatment.—The treatment of the underlying disease is of the greatest importance in all cases where any aural affection, any removable compressing swelling, as of the parotid, or syphilis, lies at the bottom of the trouble. The methods of treatment indicated in such cases are self-evident. In fresh rheumatic facial paralysis it is advisable at first to give a few doses of aspirin or antipyrin, etc. In other cases electricity is the only remedy which can give positive results, although we must not overestimate its efficiency. In the earlier stages of the paralysis we may recommend the stabile conduction of a weak constant current through the auriculo-mastoid fossæ, four to six times a week for two or three minutes—at first the anode, then the cathode, to the affected side. Later on the chief treatment is peripheral galvanization, or eventually faradization of the muscles. We place the anode in the auricular fossa and slowly stroke the different nerve branches and the muscles with the cathode. We can often confirm the fact, immediately after each sitting, that the eye closes better after galvanization of the orbicularis. Faradization excites a reflex irritation of the nerves from irritation of the skin, and hence is perhaps of service. Internal remedies (strychnin, etc.) are probably quite useless. [In old and incurable cases much benefit can be obtained by suturing the facial nerve to the lingual or spinal accessory. In this way much motion may be regained in the paralyzed muscles.—K.]

In secondary contractures we may obtain some favorable results by methodical stretching of the muscles by wooden wedges inserted under the cheeks and by massage.

4. PARALYSES IN THE REGION OF THE MUSCLES OF THE SHOULDER

Isolated paralyses of these muscles are rare, with the exception of the practically important paralysis of the serratus. Disturbances in their functions are more frequent, as one symptom in complicated paralytic states, especially in progressive muscular atrophy, and in diseases in the neighborhood of the cervical segment of the spinal cord or of the upper cervical vertebræ. In syringomyelia paralyses in the distribution of the accessory nerve are not uncommon.

Paralysis of the Sterno-cleido-mastoid (*Spinal Accessory Nerve*).—The chin is somewhat raised and turned toward the affected side in consequence of the antagonistic contracture of the other sterno-mastoid. Motion in the opposite direction is difficult. In bilateral paralysis of this muscle it is very difficult to turn the head with the chin raised, and it can be done only imperfectly. The head readily falls backward and, when the patient is lying down, it can be lifted only with the greatest difficulty if at all.

Paralysis of the Trapezius (*Spinal Accessory Nerve*).—The shoulder sinks downward and forward so that the supraclavicular fossa becomes deeper. The median border of the scapula moves outward and is not parallel to the vertebral column, as under normal conditions, but it runs obliquely from below and inward, upward, and outward. Voluntary raising of the shoulder, "shrugging the shoulder," is impaired, and it can be done only by the levator scapulæ. The arm sinks down from its own weight, and there is often pain in the shoulder from stretching the ligaments of the joint. If the clavicular portion is paralyzed, the shoulder does not move on deep breathing. Raising the arm above the horizontal is also affected, from the impaired fixation of the scapula.

Paralysis of the Pectoralis Major and Minor (*Anterior Thoracic Nerves*).—Abduction of the upper arm is difficult, the hand can be placed on the shoulder of the healthy side only by the help of the anterior bundle of the deltoid, and the palms cannot be clapped together hard when the arms are stretched forward. The function of the pectoral in forcibly drawing down the arm when raised to the perpendicular (as in chopping wood, pulling down a hanging rope, climbing, etc.) is very important.

Paralysis of the Rhomboidei (*N. dorsalis scapulæ*) causes the inner edge of the scapula to be somewhat raised from the thorax while its lower angle moves outward. It becomes difficult to approximate the scapula to the vertebræ, and wholly impossible to do so if the trapezius be also paralyzed. The movements of the extended arm inward and backward are also impaired on account of the defective fixation of the scapula. Isolated paralysis of the levator anguli scapulæ causes no noticeable symptoms, but if the trapezius be also paralyzed it becomes wholly impossible to raise the scapula. Then the approximation of the scapula to the vertebral column (rhomboidei) and the raising of the scapula (levator anguli scapulæ) are completely abolished.

Paralysis of the Latissimus Dorsi (*Subscapular Nerves*).—There is no deformity when at rest, but the arm cannot be strongly adducted inward and backward or held firmly to the trunk, and the hand cannot be placed on the sacrum. The latissimus, like the pectoralis major, acts in drawing down vigorously the raised arm. Finally, when the latissimus is paralyzed, it affects the throwing back of the shoulders, as in the position of the soldier.

Paralysis of the Rotators of the Humerus Inward and Outward.—Movements of rotation at the shoulder joint are best tested when the forearm is flexed at a right angle. In paralysis of the inward rotators (especially the subscapular, innervated by the subscapular nerve), the arm, when rotated outward, cannot be brought back again to its normal position. All manipulations, too, which the paralyzed arm tries to make on the opposite side of the body, are considerably impaired. In paralysis of the outward rotators—the infraspinatus, innervated by the suprascapular nerve, and the teres minor, innervated by the axillary—rotation of the arm outward is abolished. In writing and sewing (using the needle), the paralysis causes very marked disturbance. Paralysis of the teres major (subscapular nerve) causes hardly any noticeable disturbance, since this muscle acts merely as an aid to the latissimus dorsi and pectoralis major.

Paralysis of the Serratus Anticus Major (*Paralysis of the Long Thoracic Nerve*).—This paralysis is quite common, and therefore is of practical importance. Its most frequent cause is mechanical injury of the long thoracic nerve, such as may occur especially in porters, field hands who carry sacks, soldiers in fighting, etc. We also see at times a neuritic paralysis of the serratus, coming on with pain in the shoulder without any discoverable cause, or as the result of taking cold ("rheumatic serratus paralysis"). Finally, serratus paralysis may follow acute infectious diseases, especially typhoid fever, where it is probably also of neuritic origin. We have seen serratus paralysis after acute articular rheumatism and once after gonorrhea. Bilateral paralysis of the serratus is seen as one symptom of extensive muscular atrophy, especially in juvenile muscular dystrophy. A congenital absence of both serrati also occurs with the corresponding disturbances of function.

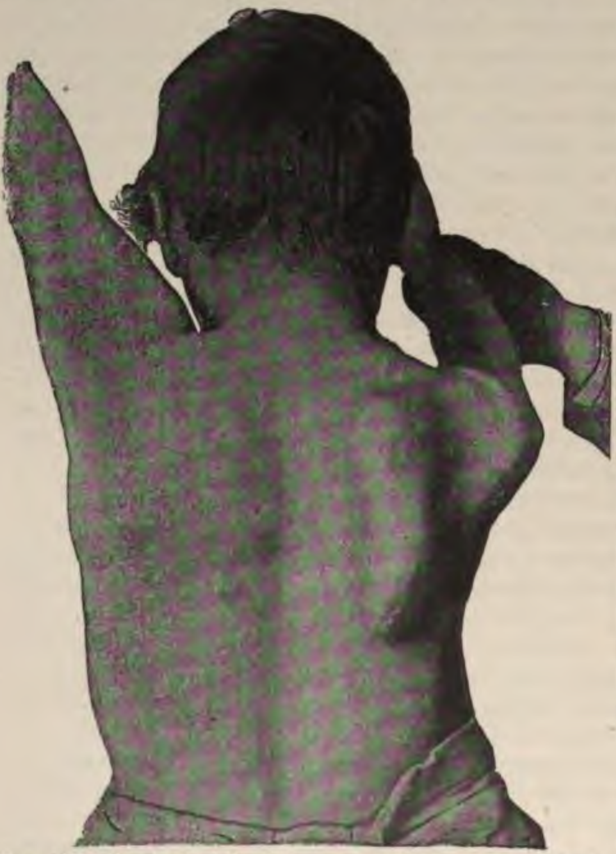

FIG. 137.—Paralysis of the right serratus (personal observation). Wing-like protrusion of the right scapula when the arm is stretched forward.

As the arm hangs down quietly, the scapula on the paralyzed side is somewhat raised and stands out a little from the chest wall, from the action of the antagonists (the traction of the levator anguli scapulæ, the trapezius, and the pectoralis), its lower angle is a little approximated to the vertebral column, and therefore its median edge runs obliquely upward and outward (traction of the rhomboidei). This faulty position of the scapula, however, is in many cases only of slight degree.

If the patient wishes to raise his arm, he can raise it only to the horizontal position, and we fail to see the projection of the tense indentations of the serratus on the lateral wall of the chest; but as soon as we seize the scapula firmly and push it forward—that is, supply the missing action of the serratus—the patient can at once raise the arm. If the arm is raised outward to the horizontal line, the scapula approaches the vertebral column. The somewhat projecting lower angle then comes almost behind the spinous process. The muscular masses of the trapezius and rhomboidei are crowded together and form a swelling which is absent on the healthy side when the arms are in the same position. If the patient moves his arm forward, which has been raised outward, the scapula turns about its vertical axis and makes a wing-like projection almost perpendicular to the surface of the back (*vide* Fig. 137). We can now take the scapula between two fingers and grasp its whole inner surface. The latter forms a deep hollow with the middle part of the back. Serratus paralysis can almost always be recognized at the first glance from this extremely characteristic and peculiar position of the scapula. In some cases the patient can still raise the arm almost completely to the perpendicular in spite of paralysis of the serratus. This is probably done by the help of the middle portion of the trapezius. Disturbances of sensation are often entirely absent, but, on careful testing, we can often find a slight disturbance in the region of the scapula and shoulder in traumatic and neuritic paralysis.

The course of serratus paralysis is usually quite tedious, but, as a rule, in ordinary peripheral paralyses, recovery takes place after several months. Many cases are incurable. Treatment consists chiefly in the application of electricity to the paralyzed nerve and muscles.

5. PARALYSES OF THE TRUNK MUSCLES

(Back Muscles, Abdominal Muscles, Respiratory Muscles)

The muscles of the back and abdomen are rarely paralyzed except as a complication of more extensive disease. Paralysis of these muscles is seen most frequently in the different forms of juvenile muscular dystrophy (*q. v.*). In cases of extensive poliomyelitic paralysis (acute poliomyelitis of infants and young adults), upon careful examination we not infrequently find, in addition, that the abdominal and back muscles are also affected. If the erector spinæ on both sides is atrophied and paralyzed, the vertebral column, when the patient is sitting, forms a curve with the convexity backward. The patient supports himself by putting the hands on the thighs, lest the trunk fall forward. When the trunk is bent forward it is difficult or impossible to regain the upright position. On standing, the trunk sinks backward, so that a plummet dropped from the upper dorsal vertebræ falls behind the sacrum. The anterior portion of the pelvis is raised by the contraction of the abdominal muscles necessary to maintain the equilibrium. In unilateral paralysis of the long muscles of the back the spine becomes scoliotic, the concavity being directed toward the healthy side. The patients do not lie straight in bed, but generally with the trunk curved. If the abdominal muscles are paralyzed, the patient can no longer raise the trunk to an erect position, when lying on his back, without the aid of the arms. If the hand is placed on the abdomen while this is attempted, no tension of the abdominal muscles can be felt. Similarly, lateral turning motions of the trunk (changing from the back to the side) become difficult or entirely impossible. Forced movements of expiration (coughing, singing, screaming) cannot be executed. On standing erect, the belly is very prominent, the anterior portion of the pelvis is sunk, and the spinal column is bent in marked lordosis. A plummet dropped from the upper dorsal vertebræ would strike about in the middle of the sacrum. In unilateral

paralysis of the abdominal muscles, the contour of the abdomen is occasionally altered, and this becomes particularly pronounced in sitting, coughing, pressing, etc. The navel is drawn toward the healthy side. Paralysis of the smaller spinal muscles, not mentioned here, is difficult to determine positively. If the extensors of the back as well as the abdominal muscles are paralyzed, it is no longer possible to keep the trunk erect.

The condition of the respiratory muscles (intercostals, diaphragm) must be also observed in all extensive paralyses, especially since it is often of great importance in regard to the prognosis. Paralysis of the diaphragm (*vide infra*) is recognized by the absence of the inspiratory protrusion of the epigastrium; paralysis of the intercostals by the absence of the inspiratory elevation of the ribs.

6. PARALYSES IN THE REGION OF THE UPPER EXTREMITY

Paralysis of the Deltoid Muscle (*Axillary* [*Circumflex*] *Nerve*).—Deltoid paralysis occurs either as one symptom of complicated paralyses involving the brachial plexus, or as an isolated traumatic and rheumatic paralysis; that is, neuritis, beginning with pains in the region of the shoulder. After acute articular rheumatism (*q. v.*), when the shoulder has been involved, we have also repeatedly seen deltoid paralysis ("arthritic or arthropathic paralysis") with atrophy of the muscles. Finally, deltoid paralysis is a common symptom in the most varied spinal and cerebral diseases. The characteristic symptom of deltoid paralysis is the impossibility of raising the upper arm when it is hanging down vertically, but we must remember that the different parts of the deltoid need not always be equally affected. The anterior bundle of the muscle raises the arm obliquely forward and inward, the middle bundle outward, and the posterior bundle backward; the latter also serves chiefly to draw the arm outward and backward when it has been raised forward. The arm is raised forward and outward by the action of the deltoid only up to the horizontal, and it is raised backward only to an angle of about forty-five degrees. To raise the arm completely to the perpendicular the serratus anticus must act (*vide supra*). If the anterior bundle of the deltoid be chiefly paralyzed, the patient can no longer put his hand to his head, raise the arm to the opposite shoulder, etc. If the posterior bundle be paralyzed he can no longer put his hand in his trousers' pocket. In complete paralysis the arm usually hangs down loosely. If the patient, nevertheless, makes a vigorous effort to raise his arm, the shoulder is usually raised and the scapula turned from the contraction of the trapezius and serratus. In a few cases only has it been observed that, where the deltoid is wholly paralyzed, the patient can still raise his arm by the aid of the upper portion of the pectoralis major or of the supraspinatus and trapezius. It is easy to avoid mistaking anchylosis of the shoulder joint for deltoid paralysis by making a few passive movements of the arm.

Paralysis of the biceps and brachialis anticus (*musculo-cutaneous nerve*) is only exceptionally an isolated paralysis, but it is quite often seen combined with other paralyses. The forearm, when in supination, cannot be flexed, but in pronation the supinator longus can still display its action of flexion. The action of supination by the biceps, which it exerts when the forearm is flexed,

is also absent. We can readily recognize this action on ourselves by making a rapid and forcible movement of supination when the forearm is flexed and then feeling the contraction of the biceps with the fingers. We sometimes see at the same time a disturbance of sensibility on the radial side of the forearm from an affection of a cutaneous branch of the musculo-cutaneous nerve.

Radial [Musculo-spiral] Paralysis.—The anatomical course of the radial nerve causes pressure paralysis of this nerve to be among the commonest peripheral paralyses. It is seen especially when the nerve is pressed against the humerus during sleep by the body or head lying on it, in drunkenness, sleeping on a wooden bench, on the hard ground, etc. The paralysis is usually noticed immediately on waking. Other traumatic influences, direct injuries of the nerve, compression in dislocation of the shoulder, in fractures of the humerus, in pressure from crutches, in bandaging or fettering the arm, etc., are also frequent causes of radial paralysis. It is worthy of note that paralysis of the extensor digitorum has been repeatedly seen to follow subcutaneous injections of ether on the extensor side of the forearm. Exposure to cold (rheumatic radial paralysis) plays no very great part in the ætiology of paralysis. Radial paralysis is also rare after acute infectious diseases. Lead paralysis, which is localized chiefly in the distribution of the radial, is discussed below.

The radial innervates the triceps and the muscles on the extensor side of the forearm. Paralysis of the triceps is present only in the cases where the point of lesion is situated quite high up, as in crutch paralyses, dislocation paralyses, plexus paralyses, etc., but it is absent, or at least it is only faintly manifest, in most of the ordinary pressure paralyses, in which the place where the radial turns about the humerus is the point of compression. Triceps paralysis is readily recognized by the impossibility of extending the forearm, but we must always make the experiment with the upper arm raised, so as to exclude the action of gravity in extending the forearm.

Paralysis of the muscles on the extensor side of the forearm may at once be recognized, since the hand hangs down relaxed in a flexed position (see Fig. 138). Any dorsal extension by the extensor carpi ulnaris and the extensores carpi radialis longus et brevis is impossible, and the lateral movements of the hand in abduction and adduction are rendered difficult. The fingers are flexed, the first phalanx cannot be extended by the extensor communis digitorum, extensor indicis, and extensor minimi digiti; but if the first phalanges be extended passively and supported, the extension of the terminal phalanges is perfectly normal, from the action of the interossei which are supplied by the ulnar nerve. The thumb is flexed and adducted, and can neither be abducted nor extended actively (extensores ossis metacarpi et primi et secundi internodii pollicis). If the forearm be extended and pronated it cannot be

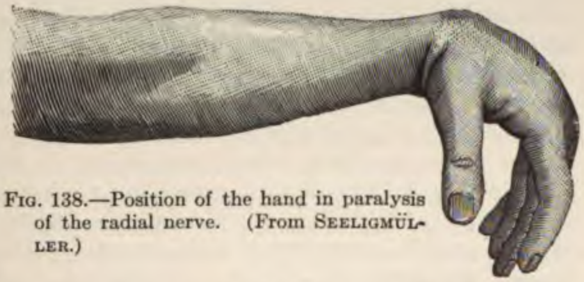

FIG. 138.—Position of the hand in paralysis of the radial nerve. (From SEELIGMÜLLER.)

paralysis of the abdominal muscles, the contour of the abdomen is occasionally altered, and this becomes particularly pronounced in sitting, coughing, pressing, etc. The navel is drawn toward the healthy side. Paralysis of the smaller spinal muscles, not mentioned here, is difficult to determine positively. If the extensors of the back as well as the abdominal muscles are paralyzed, it is no longer possible to keep the trunk erect.

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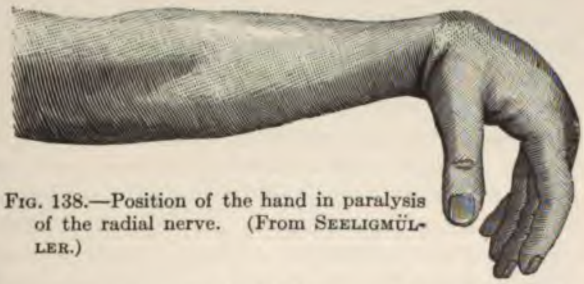

FIG. 138.—Position of the hand in paralysis of the radial nerve. (From SEELIGMÜLLER.)

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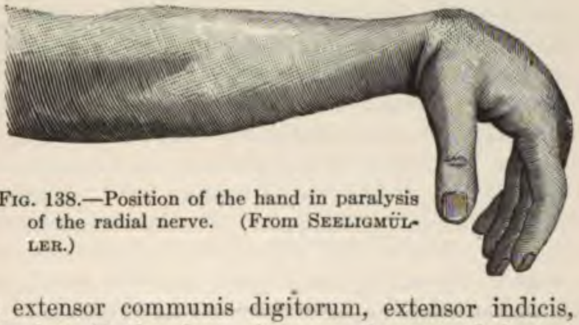

FIG. 138.—Position of the hand in paralysis of the radial nerve. (From SEELIGMÜLLER.)

supinated (supinator brevis), but the flexed forearm can be supinated by the biceps. Flexion of the forearm in supination, which is done by the biceps and brachialis anticus, is retained, but flexion when half pronated ("middle position") is weakened, from the paralysis of the supinator longus. If we have the patient make short and rapid movements of flexion of the forearm in this position we do not see the characteristic normal prominence of the tense supinator longus. The very characteristic prominence of this muscle is also wanting if the patient tries to hold his pronated and semiflexed forearm firm against forcible attempts at extension. The supinator longus is only exceptionally spared in radial paralysis.

The functional disturbance of the hand in radial paralysis is very considerable. The action of the flexors is also weakened, since their points of insertion are approximated, on account of the constant drooping of the hand. We often see, too, sensory disturbances, as well as motor, in the distribution of the radial, but these are usually slight. The chief seat is on the radial half of the back of the hand and the dorsal surface of the thumb and index and middle fingers (compare Fig. 119, page 160). The electrical excitability of the paralyzed parts corresponds to the laws that generally obtain. At the onset, and in mild cases, it is normal; at a later period, in severe cases, there are pronounced atrophy and reaction of degeneration. It is worthy of note that in all forms of radial paralysis, especially in lead paralysis, we very often find a peculiar chronic thickening and swelling of the tendons on the back of the hand, the chief cause of which is probably the mechanical tension of the tendons.

Ulnar Paralysis.—Except from the frequent implication of the muscles supplied by the ulnar nerve in extensive paralyses and atrophies, especially in progressive muscular atrophy, ulnar paralysis arises chiefly from traumatic influences, pressure, wounds, fractures of the humerus, especially fractures of the internal condyle, dislocations of the shoulder joint, etc. Primary neuritic paralyses or paralyses following acute diseases, such as typhoid fever, are much more rare.

Flexion of the hand, and especially its lateral movement to the ulnar side, is disturbed (flexor carpi ulnaris). Flexion of the last three fingers is imperfect from partial paresis of the flexor profundus digitorum, and the little finger cannot be moved at all (hypothenar muscles). Paralysis of the interossei and the last two lumbricales is most striking, by which flexion of the proximal phalanges and extension of the terminal phalanges of the fingers becomes impossible. The defective extension of the terminal phalanges is most plainly seen when we hold the proximal phalanges firmly and ask the patient to extend the fingers fully. The last two fingers remain less extended than the index and middle fingers, whose lumbricales are innervated by the median nerve. Spreading the fingers, and still more bringing them together again, are also much impaired (interossei, lumbricales). The thumb cannot be adducted against the metacarpal bone of the index finger (adductor pollicis) or held firmly in this position.

In almost all old cases of ulnar paralysis a very characteristic position of the hand is developed, besides the muscular atrophy which is especially noticeable in the interosseal furrows of the back of the hand. By the contraction of the muscles antagonistic to the paralyzed interossei (extensor and flexor communis digitorum), the first phalanges are put in marked dorsal extension, but the terminal phalanges are flexed, so that the hand assumes an actual clawing position—"claw-like hand," *main en griffe* (see Fig. 139). A similar position of the fingers occurs as a congenital anomaly (in small children), and is probably due to defective development or complete absence of the interossei.

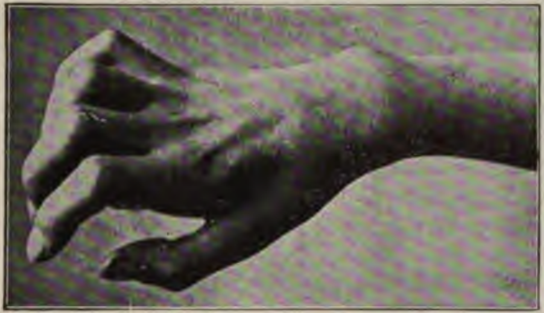

Fig. 139.—Paralysis of the ulnar nerve. Atrophy of the interossei. The terminal phalanges cannot be extended. (From the Erlangen Medical Clinic.)

The disturbance of sensibility, if it be present at all, affects the volar surface of the last two fingers, the dorsal surface of the last three fingers, and a portion of the back of the hand (see Figs. 117, 118, 119). Trophic disturbances of the skin of the fingers are not infrequently seen.

Median Paralysis.—Isolated peripheral median paralysis is seen chiefly as a traumatic paralysis, much more rarely as neuritic paralysis. Disturbances in the muscles innervated by the median are comparatively common as a complication of more extensive paralyses, in cerebral and spinal diseases (progressive muscular atrophy).

The disturbances of motion are striking. Pronation of the forearm is almost wholly abolished (pronator radii teres and quadratus). The hand can be flexed only toward the ulnar side by the flexor carpi ulnaris (paralysis of the flexor carpi radialis). The terminal phalanges of the fingers can no longer be flexed (flexor sublimis digitorum and a part of the profundus), but flexion of the primary phalanges is normal by means of the interossei. The patient can grasp an object only by the last three fingers, which can still be partly flexed by the flexor profundus digitorum (ulnar nerve). The terminal phalanx of the thumb can no longer be flexed (flexor longus pollicis), and the first phalanx can be flexed only when the thumb is also adducted by the adductor and the inner head of the flexor brevis (ulnar nerve). The thumb cannot be opposed at all (touching the terminal phalanx of the little finger with the thumb), since the muscles that act for opposition (opponens, abductor brevis, and outer head of the flexor brevis) are all innervated by the median nerve. The thumb therefore lies with the metacarpus turned backward in extension as a result of the unopposed action of the extensor longus ("ape position").

If there is any disturbance of sensibility, it is found on the volar surface of the thumb and the two adjacent fingers, and also on the dorsal surface of the terminal and middle phalanges of the index and middle fingers, and the radial side of the ring finger (see Figs. 118 and 119). In severe cases trophic disturbances, vesicles on the fingers, a shining atrophic skin, and changes in the nails are often present.

Combined Paralyses of the Arm Muscles.—Combined paralyses, in which the affected muscles belong to the distribution of several nerves, occur in various forms, especially as a result of injuries which affect the brachial plexus in the neck or shoulder—plexus paralyses. There is either direct injury of the nerves by a blow or wound, or indirect injury from luxation of the humerus, fracture of the head of the humerus and the clavicle, tumors in the supraclavicular fossa, etc. Plexus paralyses from primary neuritis also occur.

Among the many possible combinations of plexus paralyses, one form, first described by Erb, and since then repeatedly observed, deserves special mention. In this the deltoid, biceps, brachialis anticus, and supinator longus (muscles whose nerves all rise from the roots of the fifth and sixth cervical nerves) are paralyzed at the same time. The same combination of paralysis therefore occurs as if the motor roots of the fifth and sixth cervical nerves were themselves affected. The arm hangs down relaxed, and cannot be raised at all; the forearm cannot be flexed at all, but the hand and fingers have their normal mobility. The cause of the paralysis must have its seat at the point where the nerve fibers for the muscles mentioned lie near one another (see Fig. 129, page 213). The supinator brevis and infraspinatus are also not very infrequently paralyzed at the same time, so that the forearm, when pronated and hanging down, cannot be supinated, and the humerus, when rotated inward, cannot be turned outward. The supraspinatus and subscapularis may also be involved in the paralysis. Sensory disturbances are usually slight, but they may sometimes be found on the outer side of the upper arm and forearm.

This same combination of paralyzed muscles is found in a part of the obstetrical paralyses first described by Duchenne. These are sometimes seen in infants after hard labor, and are the result of injuries of the brachial plexus in turning, in bringing down the raised arm, in the Prague method, in extracting the child by the shoulders, in forceps delivery, etc. Such obstetrical paralyses may get entirely well in a few weeks or months, but not infrequently permanent atrophic paralyses remain.

Another rare form of plexus paralysis occurs from injury of the fibers coming from the eighth cervical and first dorsal nerves. This leads to paralysis of the smaller muscles of the hand (thenar, interossei) and the flexor muscles on the palmar side of the forearm. Slight disturbances of sensibility are usually present at the same time in the ulnar and median region.

In some cases of complicated paralyses of the brachial plexus, which are usually traumatic (Seeligmüller and others), coexisting symptoms on the part of the sympathetic have been observed, consisting of contraction of the pupil, narrowing of the opening of the lids, and a retraction of the eyeball on the paralyzed side. These symptoms, pointing to a paralysis of the sympathetic nerves (*vide infra*), are probably always due to a lesion of the ramus communicans of the first dorsal nerve, as follows from clinical and experimental investigations (Dejerine-Klumpke). Vasomotor symptoms in the face are usually absent, but we sometimes find a peculiar flattening of the cheeks, which has not yet been explained.

Occupation Paralyses.—In certain occupations in which certain muscles or groups of muscles or nerves are overexcited or exposed to persistent pressure, paralyses may occur which merit a brief mention. Thus, we see atrophic paralysis of the muscles of the ball of the thumb particularly in file cutters (although the toxic action of lead must also be taken into consideration), stone cutters, ironers, basket weavers, locksmiths, joiners, etc., and atrophy of the interossei in cigar-rollers. Bruns has described an isolated paralysis of the flexor pollicis longus as sometimes occurring in drummers as a result of overexertion. The prognosis of these paralyses, which are often associated with paræsthesiæ and slight sensory disturbances, is not unfavorable, provided the patient can give up his harmful calling.

General Prognosis and Treatment of the Peripheral Paralyses of the Upper Extremity.—In the prognosis of the peripheral paralyses of the arm the same points hold good that have been spoken of in the prognosis of facial paralysis. In these, too, there are mild and severe cases, the latter having complete reaction of degeneration, and a course that lasts at least several months before recovery. A number of traumatic and neuritic paralyses can be cured only up to a certain point, or they may be even entirely incurable.

The treatment can fulfill a causal indication only in comparatively rare cases, when we can succeed in removing by operation any compressing tumors, cicatrices, splinters of bone, formations of callus, etc. When nerves have been cut, suture of the nerve is an extremely important measure, which promotes recovery and which may be the only possible means to secure recovery. In fresh neuritic paralyses the well-known antirheumatics (aspirin, salicylate of soda) are sometimes used, especially if there is also pain.

In other cases the electrical treatment of paralysis promises the best success. We use the galvanic current chiefly, although we generally employ the faradic current at the same time. In regard to the method of application, we may employ the stabile action of the constant current on the very point of the lesion, especially in fresh cases, but the chief method is the electrical irritation of the paralyzed nerves and muscles. We treat the nerve above the point of the lesion in order to act in some degree against the hindrance to conduction from above and to overcome it. The muscles are irritated by galvanism by stroking the cathode over the individual paralyzed muscles. If there is reaction of degeneration, with anodic contractions predominating or exclusively present, we use the anode for the exciting pole. The other pole is placed on the back of the neck or on the seat of the lesion. Faradization of the muscles may also be of service, especially if the muscles react to faradism; but, even if this is not the case, the sensory faradic irritation has perhaps a favorable influence, since it produces a reflex irritation of the motor nerves. The single sittings should last five or ten minutes, and should take place daily or three or four times a week. The fresher the paralysis, the more favorable in general is the prognosis. Of course in such cases it is usually impossible to decide how much of the improvement is to be referred to the treatment and how much to spontaneous recovery, but we cannot deny that we sometimes obtain noteworthy success even in old and severe cases by great patience and perseverance. The treatment must in such cases be kept up for months or years, with occasional interruptions.

Embrocations with spirits and with similar substances must often be prescribed in practice, but they act favorably only when associated with methodical massage of the paralyzed muscles. We sometimes see a certain advantage, too, from local warm bathing, or from the use of the baths in Teplitz, Wiesbaden, Wildbad, etc.

7. PARALYSIS OF THE DIAPHRAGM

Isolated paralysis of the diaphragm occurs but rarely, from wounds of the phrenic nerve in the neck, also as primary or secondary neuritic (postdiphtheritic) paralysis, and finally in hysteria. Muscular paresis of the diaphragm seems to develop sometimes as a result of inflammation of the serous layer of the diaphragm. The paralysis of the diaphragm, which comes on as one symptom in more extensive paralyses, is more frequent and practically more important. In diseases of the upper cervical cord, in ascending myelitis, in compression of the cervical cord by vertebral caries or meningeal tumors, in progressive muscular atrophy, in multiple neuritis, etc., the development of paralysis of the diaphragm is the cause of the rapidly fatal termination which follows the appearance of disturbance of respiration. The root of origin of the phrenic nerve is principally the fourth cervical.

The symptoms of paralysis of the diaphragm are readily recognized, especially in the ordinary bilateral affection. We detect the modification of the respiratory movements at the first glance. While we are struck by the marked upper thoracic respiration, which becomes very labored on the slightest cause, the visible and palpable protrusion of the epigastrium on inspiration is entirely absent. Instead of this there is usually an inspiratory retraction in the epigastric region. The respiration is but little accelerated in simple paralysis of the diaphragm as long as the patient is perfectly quiet; but in some cases the development of a severe bronchitis, from the defective respiration in the lower lobes of the lungs, causes constant dyspnœa. The explanation of the bronchitis may be found in the fact that the action of abdominal pressure is very much diminished by the constant high position of the diaphragm, which may be made out by percussion, and consequently the cough and the expectoration of secretion are very imperfect.

The prognosis is favorable only in hysterical and rheumatic paralyses; otherwise it is usually very unfavorable. In regard to treatment, the only thing that can be tried is to stimulate the diaphragm from the phrenic in the neck by faradism or galvanism, while the other pole is placed on the region of the insertion of the diaphragm in the thorax. A transverse conduction of the constant current through the diaphragm, associated with changes of the current, may also have a favorable influence.

8. PARALYSES IN THE REGION OF THE LOWER EXTREMITY

Paralysis of the Crural Nerve.—Crural paralysis is but rarely isolated. It is seen after injuries, after compression of the nerve by tumors of the pelvis or thigh, in disease of the vertebræ, psoas abscess, etc. There is also a primary neuritis of the crural nerve, especially, not infrequently, a bilateral crural paralysis in alcoholic or apparently spontaneous polyneuritis; and, finally, paralysis of the quadriceps sometimes follows acute articular rheumatism and other affections involving the knee joint.

The muscles innervated by the crural nerve are the iliopsoas, the sartorius, pectineus (occasionally also supplied by the obturator nerve), and the extensor cruris quadriceps. The first three muscles mentioned, particularly the iliopsoas, are the flexors of the thigh at the hip. If they are all paralyzed, then, at most, a slight flexion of the thigh can be produced, but attempted only with the aid of the adductors. If the iliopsoas alone be paralyzed, flexion is produced with the aid of the sartorius and the tensor vaginæ femoris (inferior gluteal nerve); these muscles may then undergo a vicarious hypertrophy. Paralysis of the sartorius and pectineus does not produce very marked symptoms, but can be recognized by the absence of tension in these muscles in resisted movements. Pronounced paralysis of the flexors of the thigh makes it difficult to raise the body from the dorsal position. Paralysis of the quadriceps extensor is readily determined by testing the extension of the lower leg. If the thigh be flexed passively in bed, the dependent lower leg cannot be extended or raised in the air. In peripheral paralysis of the quadriceps the patellar reflex, of course, is absent. In paralysis of the crural nerves, walking and standing are very difficult or almost impossible, since the iliopsoas is necessary for the stride forward and the quadriceps for fixation of the knee joint. If there is any disturbance of sensibility it is found in the lower half of the anterior surface of the thigh and on the inner side of the leg down to the great toe (saphenous nerve, see Figs. 120, 121, page 160).

Paralysis of the Obturator Nerve.—Paralysis of this nerve is very rarely seen as an isolated phenomenon. The chief symptoms are the defective adduction of the thigh (the adductor magnus, longus, and brevis, and the gracilis), and the impossibility of crossing one leg over the other. Rotation of the thigh outward is also disturbed (obturator externus). Some disturbance of sensibility may be found on the inner side of the thigh.

Isolated Paralyses in the Distribution of the Gluteal Nerves.—Paralyses of these nerves are not common, but the muscles supplied by them are often very markedly implicated in more extensive paralytic conditions, especially in muscular dystrophy and multiple neuritis. Paralysis of the glutæus maximus is rendered noticeable by the fact that this muscle extends the thigh on the pelvis. It is therefore especially called into play in going upstairs, climbing mountains, and rising from a sitting or recumbent position. All these movements are rendered nearly impossible when the muscles are paralyzed. While walking on level ground is only slightly affected, the patient cannot step up upon any step; he can get up from a chair only with effort, etc. On rising from a sitting position from the floor the trunk is raised by supporting the arms on the thighs (see the illustration in the chapter on Juvenile Muscular Atrophy). When the patient is in bed we may test the function of the glutæus maximus by having him resist flexion of the thigh, or by holding the flexed thigh tightly with the hand and making the patient extend it. The glutæus medius and glutæus minimus are abductors of the thigh, and also fix the thigh on the pelvis. If they are paralyzed the patient has a very characteristic waddling gait. On account of the predominance of the adductors the feet are then advanced near each other or even one over the other. The glutæus medius is also a rotator of the thigh inward, so that when it is paralyzed the outward rotators (pyriformis, obturator internus and externus, gemelli, and quadratus femoris) exert a predominant action.

Paralyses in the Region of the Sciatic.—These paralyses come from traumatic lesions, from continuous pressure on the nerve from working in a squatting position (field work, etc.), from pelvic tumors, and in hard labors (pressure of the forceps, or, with a narrow pelvis, pressure of the presenting head on the nerves). Isolated primary neuritic paralysis in the sciatic region is rare, but we quite frequently see peroneal paralysis as a symptom of more extensive multiple neuritis (alcoholic polyneuritis, neurotic muscular atrophy, etc.). Diabetic neuritis may also be localized in the peroneal nerve. Very marked paralytic symptoms in the region of the sciatic are seen in diseases of the conus terminalis and the cauda equina (lesions of the lumbar vertebræ and sacrum).

PERONEAL PARALYSIS.—Of the two main branches of the sciatic, the peroneus and the tibial, the former is far more frequently affected than the latter. Even in injuries of the sciatic trunk, the peroneal paralysis is usually much more evident than the injury of the tibialis fibers. We can readily understand that the peroneal nerve itself is quite often subject to external injury from its superficial position. The symptoms of peroneal paralysis are very readily recognized. The flaccid drooping of the toe strikes the observer at once. On walking this becomes very marked, and the tip of the foot often sticks to the floor. The patient, therefore, has to raise the thigh higher, and to put the foot down awkwardly, toe first ("high action," "*steppage*"). Dorsal extension of the foot (tibialis anticus) and of the proximal phalanges of the toes (extensores communis digitorum longus et brevis and extensor hallucis longus), and also abduction of the foot and raising its outer border (the peronei), are almost impossible. In old cases a permanent toe drop (talipes equinus or varo-equinus) develops, usually as a result of secondary contracture of the muscles of the calf, and this is often associated with a permanent flexed position of the toes from secondary contracture of the interossei.

PARALYSIS OF THE TIBIAL NERVE makes plantar flexion of the foot impossible (gastrocnemius and soleus). The patient can no longer rise on his toes. Adduction of the foot (tibialis posticus) and plantar flexion of the toes (flexor communis digitorum and flexor hallucis longus) are also abolished. As a result of secondary contractures, talipes calcaneus sometimes develops, and also a claw-like position of the toes, with dorsal extension of the first and plantar flexion of the last phalanges from paralysis of the interossei. If the popliteal be also paralyzed, the flexed lower leg can no longer be rotated inward.

COMPLETE PARALYSIS OF THE TRUNK OF THE SCIATIC.—In this condition there is sometimes added to the symptoms mentioned the inability to flex the leg backward on the thigh (to be tested when the patient is lying on his side or standing), which is due to a paralysis of the biceps, semimembranosus, and semitendinosus. In unilateral paralysis of the sciatic, walking is still possible, since the leg is fixed at the knee by the quadriceps extensor and is rigid like a wooden leg.

The distribution of the disturbance of sensibility on the posterior surface of the whole leg is given in Fig. 120. Vasomotor and trophic disturbances, cyanosis, coldness and atrophy of the skin, and changes in the nails are often present. Atrophy and the electrical reactions of the paralyzed muscles occur under the same conditions as in all other peripheral paralyses.

Paralyses from diseases of the cauda equina will be described later in the chapters on the Pathology of the Spinal Cord.

Treatment of all the paralyses last described follows the same rules as are given for the management of peripheral paralyses in the upper extremity.

9. TOXIC PARALYSES

Lead Paralysis.—Of all the toxic paralyses, that from lead poisoning is practically the most important. It is a common symptom of chronic lead poisoning, and is seen chiefly in those persons whose occupation gives rise to a long-continued absorption of small amounts of lead, especially, therefore, in typesetters, typecutters, and typefounders; in artists and house painters, from lead colors; in potters, from lead glaze; in tinkers, file cutters, etc. Lead paralysis may occur as the only symptom of chronic lead poisoning, or it may be associated in various ways with other symptoms (lead colic, lead encephalopathy, etc.).

The investigations of Leyden, Zunker, Schultze, and others agree in their main conclusions that the anatomical cause of lead paralysis is certainly a degenerative disease and ultimate atrophy of the peripheral motor nerve fibers belonging to the paralyzed muscles, caused by the toxic influence of lead. The atrophy of the muscles is therefore to be regarded principally as a purely secondary disease, although it is possible that the muscles may also be injured by the lead at the same time with the nerves. The spinal cord, and especially the motor ganglion cells in the anterior horns of the gray matter, remain, as a rule, unaltered, but undoubtedly, in severe, incurable cases, there may also finally develop a toxic atrophy of these ganglion cells.

Lead paralysis shows an extremely typical localization in the great majority of cases, and it affects by far the most frequently a part of the radial distribution. A paralysis of the extensor communis digitorum comes on rapidly or slowly. Extension of the proximal phalanx of the middle and ring, and later of the index and little fingers, becomes impossible, but the extension of the terminal phalanges by the interossei remains normal. There often follows later a paralysis of the extensor and abductor longus pollicis and the extensors of the wrist, and in severe cases a paralysis of the interossei and thenar muscles, while the supinator longus and the triceps almost always remain free in a remarkable way. In much rarer cases lead paralysis affects the deltoid, biceps, brachialis anticus, and supinators. In comparing a large number of occupation lead palsies, it was demonstrated that, as a rule, those muscles which were principally used were most markedly affected (Teleky). Paralysis of the lower extremities is also very rare. It has been observed principally in children. We have seen a very characteristic case in which, besides extensive paralysis of both arms, walking was for some time almost impossible on account of bilateral paralysis of the iliopsoas and extensor cruris (crural nerve). The patient recovered completely. Other observers have seen peroneal paralysis from lead poisoning [sometimes without noticeable involvement of the arm muscles.—K.]. Paralysis of the laryngeal muscles has also been described.

Lead paralysis is usually bilateral. Ordinarily the right forearm is first affected and the left arm some days or weeks later. In all severe cases a pronounced atrophy and electrical reaction of degeneration develop in the paralyzed muscles. It is an interesting point that changes in the electrical excitability (slow galvanic contractions, etc.) may sometimes be made out in muscles which can be perfectly well moved by the will (see page 219). The sensibility is almost invariably normal, or at most only very slightly altered, so that the sensory nerves are manifestly unaffected by the lead.

Lead paralysis permits a favorable prognosis in the cases where the patient can be removed from the injurious influence of the poison. Recovery takes place after several weeks, or in severe cases after some months. Relapses of paralysis and complications with other morbid results of chronic lead poisoning, however, are, of course, frequent.

The treatment is the same as in all other peripheral paralyses. Electricity is first to be considered. Local sulphur baths and the internal use of iodid of potassium are also recommended.

[In the severer forms of chronic lead poisoning the central nervous system may be affected as well as the peripheral nerves. In the chapter on poisons (*q. v.*) brief reference has been made to some of the other conditions due to lead poisoning. Besides lead colic and lead arthralgia various forms of anæsthesia and paræsthesia may occur. In the forms of lead encephalopathy, besides the general symptoms of headache, mental unrest, and convulsions referred to above, we sometimes meet with symptoms suggestive of a localized lesion, monoplegia or hemiplegia, hemianopsia, amaurosis, etc. In other cases the existence of progressive dementia with tremors, convulsions, etc., may lead to a mistaken diagnosis of general paralysis. In other cases the symptoms are suggestive of combined sclerosis of the spinal cord—ataxic paraplegia (*q. v.*). In cases of doubtful diagnosis it may be of assistance to examine the urine for lead, after iodid of potassium has been given in 5- to 10-gr. doses thrice daily for a week. The method of making such an examination will be found in the treatises on urinary analysis. If large doses of iodid be given there is some danger that the lead will be liberated too rapidly and that more acute symptoms may ensue. J. J. Putnam has shown that lead is sometimes found in the urine of healthy persons. The pathology of chronic lead poisoning is still undetermined. The cerebral symptoms have been attributed to disseminated foci of encephalitis or to more chronic degenerative processes. Since lead poisoning may cause contracted kidney, the possibility of uræmia is also to be considered. Lead has been found in the urine in various cases of chronic degeneration of the spinal cord, and it may act as a cause of such affections, especially of combined scleroses. It is still uncertain whether such degeneration is due directly to the toxic influence of the lead or to the severe anæmia produced by lead (compare the chapter on Pernicious Anæmia). In all severe cases of chronic lead poisoning iodid of potassium should be given for a long time to insure the elimination of the lead.—K.]

Arsenical Paralysis.—Arsenical paralysis is rare in chronic arsenical poisoning (colors containing arsenic, hangings, etc.), but it is much commoner after acute poisoning. After the first violent gastrointestinal symptoms are over, a severe form of nervous disease manifests itself either immediately or two or three weeks later. The chief symptom of this affection is an extensive atrophic paralysis of the arms and legs. The legs are often affected alone, or at least the trouble is much more pronounced in them. The muscles of the trunk may also be involved in the paralysis. In general, the rule holds in arsenical paralysis also that the extensor muscles are more affected than the flexors. Electrical reaction of degeneration is found in the atrophied muscles. The accompanying disturbances of sensibility are also very characteristic, either anæsthesia, or especially paræsthesia and severe pains in the sacrum, arms, and legs. The nerve trunks are sometimes very sensitive to direct pressure. Ataxic disturbances, similar to those in alcoholic neuritis, also occur as well as paralysis. Trophic disturbances in the nails, hair, etc., have been repeatedly observed.

There are only a few investigations in man as to the anatomical cause of arsenical paralysis, but there can hardly be a doubt that the chief disturbance is a severe neuritic change. All the morbid symptoms are in harmony with this (see the chapter on Multiple Neuritis) as well as the general course of the disease, which in most cases ends favorably. Of course we must not exclude the fact that both in lead and in arsenical paralysis the cells in the anterior horns may also be affected in some severe cases.

Of the other less frequent toxic paralyses we may mention here phosphorus paralysis, copper and zinc paralysis, and mercurial paralysis, which in a few cases has been seen after the therapeutic use of mercury. Polyneuritis must be considered to be the chief lesion in all these paralyses. Very peculiar nervous disturbances associated with paralysis occur in manganese grinders from chronic manganese poisoning. The clinical picture in many ways resembles that of multiple cerebro-spinal sclerosis. Of organic compounds, carbonic oxid and bisulphid of carbon may lead to toxic paralyses. Details as to the important alcoholic paralysis will be found in the chapter on Multiple Neuritis.

CHAPTER III

THE DIFFERENT FORMS OF LOCALIZED SPASM

1. SPASM IN THE MOTOR DISTRIBUTION OF THE TRIGEMINUS

Tonic spasm of the muscles of mastication is called trismus. As an independent disease it is very rare, but it often occurs as one symptom in complicated forms of spasm and other nervous affections, as in tetanus, in the epileptic attack, in hysteria, meningitis, etc. Both jaws are pressed firmly together, and we can feel through the cheeks the hard and tense masseters. In unilateral spasm of the pterygoids the lower jaw is pushed laterally in the opposite direction.

Clonic spasm of the muscles of mastication—masticatory facial spasm—consists of a paroxysmal and constant movement of the lower jaw, almost always in a vertical, but rarely in a horizontal direction. The single movements follow one another usually in a regular, rapid rhythm, and produce an audible chattering of the teeth. The mucous membrane of the mouth and the tongue is rarely injured.

The cause of these spasms cannot always be established. Sometimes they seem to be of reflex origin, as in affections of the lower jaw, the teeth, or even of distant parts. We once saw a case which lasted for years which was caused by a severe fright, and which therefore must be regarded as " hysslightly altered, so that the sensory nerves are manifestly unaffected by the lead.

Lead paralysis permits a favorable prognosis in the cases where the patient can be removed from the injurious influence of the poison. Recovery takes place after several weeks, or in severe cases after some months. Relapses of paralysis and complications with other morbid results of chronic lead poisoning, however, are, of course, frequent.

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The cause of these spasms cannot always be established. Sometimes they seem to be of reflex origin, as in affections of the lower jaw, the teeth, or even of distant parts. We once saw a case which lasted for years which was caused by a severe fright, and which therefore must be regarded as "hysterical," and also a case of clonic spasm of the masseters and mylohyoids of hysterical origin in a ten-year-old boy.

The *treatment*, apart from the treatment of the primary affection, must aim at removing the cause of the disease, if there be one, such as decayed teeth. In other respects electricity is of value in many cases, applied either by passing the constant current through the muscles, by faradizing them, or by using the wire brush. Of internal remedies we may try narcotics, bromid of potassium, atropin, etc.

Artificial feeding is of great importance, if the patient cannot take food voluntarily from a persistent trismus. It is best to introduce a small œsophageal tube through the nose into the œsophagus. Rectal feeding is insufficient for a permanent method, but still it is sometimes of service. In some cases a successful attempt has been made to overcome the closure of the jaws gradually by pushing wooden wedges between the teeth.

2. CLONIC FACIAL SPASM

(Mimetic Facial Spasm. Facial Tic. Convulsive Tic)

We know little that is definite as to the *ætiology* of facial spasm, the most frequent and practically the most important form of isolated spasm. In some cases, perhaps, the disease is to be referred to a lesion of the trunk of the facial, from exposure to cold, aural affections, or disturbances at the base of the skull (such as aneurism of the vertebral artery pressing on the trunk of the facial, as was seen in one case), or else to a reflex irritation of the nerve, as in trigeminal neuralgia, carious teeth, painful affections of the eyes, etc. It may be that many cases are not of peripheral but of central origin, from the facial center in the cerebral cortex. The disease may also appear after violent mental excitement. Finally, imitation and the habit of grimacing play a part in many cases, especially in children, that is not to be underestimated (hysterical facial spasm). At any rate, we may see from what has been said that we should not judge all cases of mimetic facial spasm in the same way, but it is the most characteristic cases, due apparently to organic causes, which usually we are wholly unable to explain. Repeated observations have established the fact that the disposition to the disease is heightened by a general hereditary neuropathic taint. The tendency to twitch the facial muscles is very often noticed in nervous and irritable persons, but there is an essential difference between this grimacing and the true facial spasm with its lightning-like muscular contractions.

The *symptoms* of true convulsive tic consist of alternating, short, lightning-like contractions in almost all the muscles supplied by the facial. The disease is usually unilateral, often extending over the whole distribution of the facial, but sometimes confined only to individual parts—partial facial spasm. Sometimes the contractions are almost constant, though varying in intensity, so that the patient involuntarily makes the strangest faces; but they often appear in separate attacks, which last but a short time, and are interrupted by completely free intervals. The attacks arise without any special cause, or they may be excited by talking, voluntary movements, sensory and mental impressions, etc. In some very severe cases the contractions also invade the neighboring territory—the muscles of mastication, the tongue, the muscles of the neck, and even the upper arm. Voluntary motion in the muscles is perfectly normal, except for the disturbing influence of the spasmodic movements. All sensory disturbances are also lacking; there is neither anæsthesia nor pain.

A common partial form of facial spasm, which is entirely or almost entirely isolated, deserves special mention—blepharospasm, or spasm of the eyelids—that is, a tonic or clonic spasm in the orbicularis palpebrarum. The tonic form may be of reflex origin in affections of the eye, but sometimes it is excited from other regions of the trigeminus. It is, as a rule, bilateral, and it may last for days or weeks, sometimes with occasional interruptions. Graefe first noticed that the spasm could sometimes be instantly checked by pressure on the points of exit of the branches of the trigeminus, and often by pressure upon certain parts of the vertebral column or other parts of the body, so that the eyelids "fly up as if by a spring." I have no doubt that in such cases we usually have to do with conditions of hysterical spasm. Clonic spasm of the lids, nictitating spasm, consists of a spasmodic winking and contraction of the eyes, which is sometimes almost constant. Here, too, we can often make out a reflex origin for the spasm, but frequently we cannot find any cause.

Facial spasm, in its severe forms, and especially blepharospasm, is always a troublesome disease for the patient, and causes very much disturbance. The course is often very tedious. Sometimes there are long intervals, as we have seen during pregnancy, and then the spasm begins anew. In not very rare cases the affection becomes habitual and lasts for life.

Treatment is, therefore, usually a difficult and thankless task. The best results may be obtained where we can succeed in removing any reflex cause of the spasm, as in extracting decayed teeth, treating affections of the eye, or, in some cases, resecting the supraorbital nerve. In reflex blepharospasm it is sometimes beneficial to put a few drops of cocain solution into the eye. In the electrical treatment of facial spasm our chief attention must be directed to any pressure points, to which we make a stabile application of the anode of the constant current. If there are no pressure points, we put the anode on the trunk of the facial and the different branches of the pes anserinus. In cases of reflex origin Berger obtained good results by applying the anode to the occiput, just under the protuberance, while the cathode was held in the hand—galvanization of the medulla. The single sitting should last five or ten minutes. The faradic current—a slowly "swelling current"[1] —has also been recommended. Of internal remedies we should first try bromid of potassium, and then arsenic, atropin, curare, or oxid of zinc. The action of these drugs is always very uncertain. A favorable result, however, has been obtained by nerve stretching, in a part of the cases operated on, at least so far that the ensuing paralysis troubles the patient less than the constant twitching. When the paralysis ceases the twitchings usually begin again, but in some cases the benefit has been permanent. Finally, we must mention that the use of the red-hot iron has sometimes been followed by a decided improvement of the spasm in old cases. We employ cauterization along the cervical vertebræ, on the trunk of the nerve, or on any existing pressure points, by means of Paquelin's thermocautery.

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[1] [A current that begins weak, and is gradually made stronger and then weaker.—K.]

In the cases which are not to be regarded as a form of organic spasm, but are to be classed as hysterical, due to bad habits, etc., a methodical training of the will by gymnastic exercises may be attended with the best results.

[A distinction may well be made between facial and other forms of spasm and tic. Spasm is a motor reaction consecutive to pathological irritation at any point on a bulbo-spinal reflex arc, and is usually limited to the domain of a single motor nerve. A tic is a coördinated, systematized, purposive act, often involving the action of muscles supplied by various nerves. In a true tic the movement never injures the patient, as the uncoördinated spasm of chorea may do. It usually develops in neuropathic subjects with tendencies to various forms of obsession, and may often be regarded as the motor reaction to some obsessive idea, often of a defensive character. Tics may therefore often be regarded as manifestations of a psychasthenic condition (see the chapter on Psychasthenia) rather than of hysteria. In such cases very much benefit can be obtained by intelligent gymnastic treatment in the way of muscular reëducation as proposed by Meige and Feindel. In cases of true facial spasm, or of spasm in the distribution of other nerves, relief may be obtained from injections of 2 or 3 drachms (5 to 10 c.c.) of seventy-per-cent alcohol with a little cocain into the sheath.—K.]

3. SPASM IN THE REGION OF THE HYPOGLOSSAL NERVE. LINGUAL SPASM

Although the tongue is often implicated in complicated forms of spasm, such as hysterical or epileptic spasms, isolated spasms in it are very rarely seen. They are relatively most common in hysteria. They occur, however, either in a clonic or a tonic form, and then they cause a marked disturbance of speech, or even of respiration, if the spasm draw the tongue backward. In the latter case it may be necessary to use inhalations of chloroform, and to draw the tongue forward by force.

4. SPASMS IN THE MUSCLES OF THE NECK

Tonic and clonic spasms in the region of the muscles of the neck are not very frequent, but they appear in very various forms, and are at times a very severe and persistent affection. We can usually discover nothing definite as to the ætiology of this condition. When a demonstrable organic lesion of the brain, the cervical vertebræ, etc., is the cause of the spasm, we are not dealing with a true tic, but with a symptomatic irritative condition, which has an entirely different significance. A reflex origin of spasm is, as a rule, also not demonstrable. Recently, however, several authors (H. Curschmann and others) have called attention to the possibility of a primary affection of the labyrinth. Colds, traumata, toxic influences, preceding acute infections, etc., can occasionally be demonstrated as exciting factors; they do not, however, possess any decided ætiological significance. The true tic is generally a disease which arises on the basis of a general neuropathic constitution. Almost all cases which I have seen occurred in nervous persons who were easily excited mentally, and came from nervous families. Tics of the muscles of the throat and neck belong to the same group of psychomotor neuroses as the true facial tic—that is, a condition in which morbid, unconscious, and forced central (cortical) motor stimuli keep the affected muscles in a continuous state of tonic-clonic spasm. This disease is closely associated with myoclonia and certain hysterical convulsive spasms. The nomenclature is not as essential as a correct conception (as far as possible at the present time) of the nature of this group of diseases.

Although the spasms (*vide* Figs. 140 and 141) are rarely strictly limited to a definite group of muscles or the distribution of a single nerve, we can, nevertheless, as a rule, indicate certain muscles as being chiefly affected. Most frequently the muscles innervated by the spinal accessory nerve (sterno-cleido-mastoid and trapezius) are affected. We speak of this as a spinal accessory spasm. Through the contraction of the sternomastoid, the head is turned

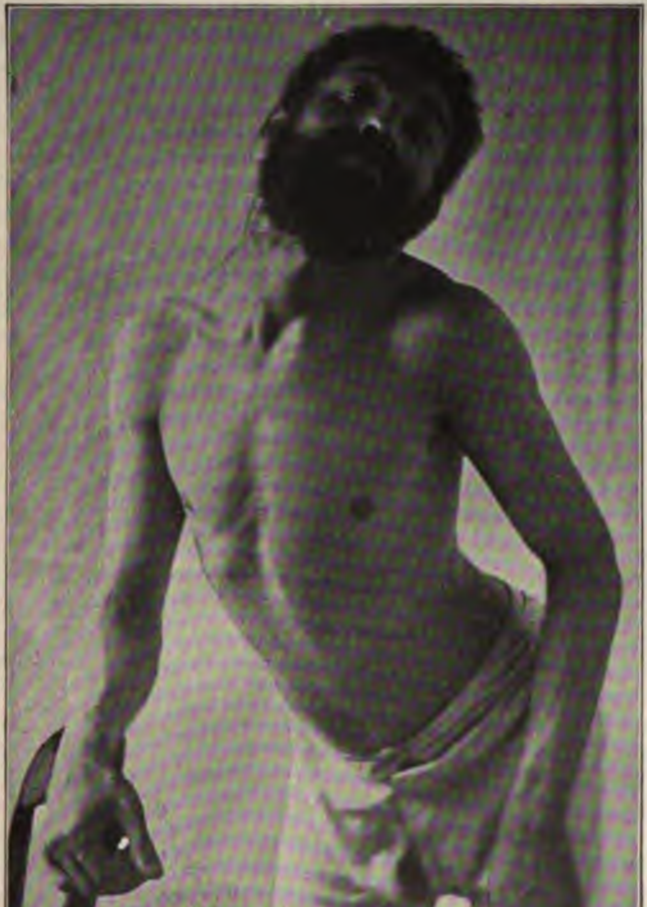


FIG. 140.—Severe case of spasm of the accessory nerve (trapezius and sterno-mastoid muscles). (From the Erlangen Medical Clinic.)

toward the opposite side, and the chin raised slightly. Spasm of the trapezius causes the head to be drawn backward on the shoulders. Generally the spastic positions of the head alternate, so that a condition of continuous unrest prevails. By placing the fingers on the muscles, we can determine (though of course not very readily) which muscles contract convulsively. Not infrequently the splenius muscle is also affected by the spasm, and the head is then drawn backward and toward the affected side. Probably the deeper small muscles of the head and neck also take part in the spasm, although this can never be directly demonstrated with certainty. In severe cases, however, an extension of the spasm to the muscles of the shoulder and upper arm (pectoralis, latissimus, *et al.*) has been observed so that the arm and the entire trunk assume a peculiar position. Pure rotatory movements of the head, apparently produced by contractions of the obliquus capitis muscle, are known as *tic rotatoire*.

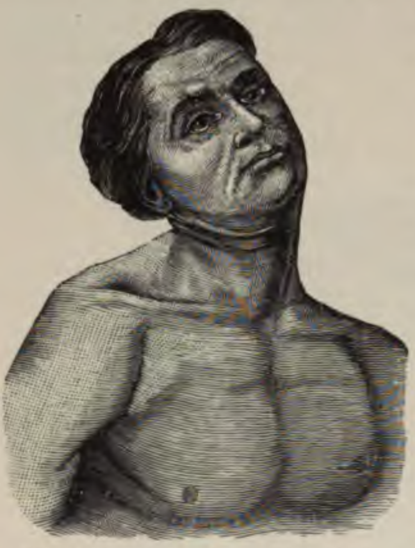


FIG. 141.—Spasm of the right splenius capitis. (From DUCHENNE.)

The *course* of the forms of spasm described is usually very tedious. There are mild cases which recover in a few weeks or months, but, on the other hand, many of these forms of spasm develop into a very distressing, habitual, chronic affection. In these cases any steady occupation, such as reading, writing, or manual labor, is rendered almost impossible by the constantly recurring spasmodic lateral and rotatory movements of the head. Any mental excitement, a feeling that he is noticed, etc., increase the patient's spasms, while they become milder when he is completely at ease. During sleep the spasms generally (*vide infra*) cease entirely. Supporting the head slightly also frequently affords relief. Many patients hold their head with their hands continually. In some cases the twitching ceases entirely when the body is wholly at rest, but it returns at once on any voluntary movement. The condition of general mental excitement has been referred to above. The occurrence of mental disturbances (melancholia, confusional insanity) has been repeatedly seen, which is not at all remarkable, considering the general nervous constitutional basis of the disease. The patient's general condition may finally become very poor. Many fall into the morphin habit.

The *prognosis* should be always guarded. As has been already stated, the disease may persist for years, in spite of all attempts at treatment. But, on the other hand, even after long duration, cures, or at least great improvement, frequently occur.

The *treatment* of these chronic spasms is always a difficult and fatiguing problem. Accepting the assumption of a constitutional basis, general constitutional measures (physical and mental rest, baths, fresh air, tonics, etc.) must be considered first. The local treatment should come next—i. e., the suppression of the morbid innervation and the restoration of the normal muscular balance. The most rational of all treatments are methodical exercises—that is, the slow practicing of regulated innervations. With perseverance, skill, and patience much can be accomplished. Further therapeutic measures, which have probably a psychical suggestive influence, are also employed. Under this category are included suitable supporting devices (corset with head rest), and occasionally even the application of firm bandages (plaster-of-Paris jacket, etc.). All mechanical devices of this description are occasionally so poorly borne, and cause so much torture to the patient, that they have to be discarded. On the other hand, I have seen some excellent results from careful orthopedic treatment. A methodical massage of the affected muscles is further recommended, and, above all, electrical treatment. The method of treatment consists of the application of the anode to the affected nerves and muscles, or of the use of a swelling faradic current, or of the faradic brush to the skin over the affected muscles. Very often we have to change the method, and we must try to find out, by testing, the most efficient way to employ electricity. Of internal remedies, narcotics, such as subcutaneous injections of morphin, are indispensable in severe cases, but we must be very careful in their use. We may also try bromid of potassium, antipyrin, arsenic, valerianate of zinc [belladonna], and other nervines. In severe cases we resort to the use of the red-hot iron to the back of the neck. I recollect a very severe case which was finally cured with the actual cautery. I have no great confidence in the many surgical procedures which have been proposed (stretching and division of the spinal accessory nerve, division of muscles and tendons); in desperate cases, however, such an attempt is justifiable.

In this place we may mention also the peculiar rhythmic spasms which occur occasionally in children during the first few years of life, and are known as spasmus nutans, salaam convulsions, nodding spasms, etc. There is a continued nodding movement of the head, occasionally also a shaking and turning, with or without involvement of the trunk muscles. Blepharospasm and nystagmus have been repeatedly seen in the same subject. Occasionally these peculiar, forced, pendulum-like movements occur only during sleep ("*tic de sommeil*"). All these symptoms are indications of a neuropathic constitution, but are generally without any serious significance. Special treatment is therefore unnecessary. The condition disappears gradually of its own accord.

5. SPASMS IN THE MUSCLES OF THE SHOULDER AND ARM

Clonic spasms in the upper extremity are rarely isolated, as in the pectoralis major, but are more frequently combined with other forms of spasm and other nervous symptoms. They sometimes seem to be of reflex origin, as in the clonic spasms associated with brachial neuralgia, and also the spasms sometimes seen in amputation stumps. For many of these forms of spasm the cause is still wholly unknown. Most of the cases are undoubtedly hysterical.

Isolated tonic spasms, in single muscles, or groups of muscles, in the upper extremity, have been repeatedly observed. Tonic spasm of the rhomboidei causes an oblique position of the scapula, so that its inner border runs obliquely upward and outward from below and inward. This makes it hard to raise the arm above the horizontal line, as in serratus paralysis, but the separation of the scapula from the chest wall, which is so very characteristic of the latter, is wanting. Tonic spasm in the levator anguli scapulæ is rare, except in connection with spasm of the rhomboidei or trapezius. In it the shoulder is raised and the head is bent a little to the affected side. Isolated tonic spasms in the pectoralis major, latissimus dorsi, deltoid, etc., are, on the whole, easy to recognize, but they are of very rare occurrence. Tonic flexor spasms of the hand and fingers are more common. We have ourselves seen several such cases, some of them lasting for months. In one case the spasm could at once be relieved by placing the anode of a moderately strong galvanic current on the median nerve. In another case the flexor spasm of the fingers had followed a mild acute inflammation of the wrist joint. In such cases, also, hysteria undoubtedly plays a part much more frequently than was formerly supposed. In tonic flexor spasms of the hand we must also think especially of tetany (*q. v.*). Brief tonic spasms of the flexors of the fingers and of the thumb muscles are of quite common occurrence. They are seen especially in persons who have to do some severe and persistent bodily work, also in alcoholic subjects, patients with gastric disease or chronic nephritis, etc. These cases do not all belong in the same category. Some are probably due to purely myopathic irritative conditions; other cases are more related to tetany (*q. v.*).

We can scarcely give any general rules for prognosis or treatment. It is always important to remove any trouble which may cause the spasm. We may also try, first of all, electricity (a stabile galvanic current, applying the anode to the affected muscles and their nerves) and methodical gymnastics, the latter especially in all cases having any relation to hysteria (*q. v.*).

6. SPASMS IN THE MUSCLES OF THE LOWER EXTREMITY

Clonic spasms in the muscles of the lower extremity are always, with rare exceptions, a symptom of spinal or cerebral disease. Of the tonic spasms the most frequent and the best known are the painful spasms in the calves, or cramps, which are apt to come on after great muscular exertion, mountain-climbing, or dancing. Some persons have an especially marked predisposition to such cramps, especially after muscular exertion (dancing, etc.). Occasionally these cramps develop very readily after making certain movements or holding the foot in a certain position. Similar painful cramps sometimes come in other muscles besides the calves, as in the abductor hallucis, etc. Constantly recurring, extensive, and painful spasms in the leg muscles may, as we have seen, form a very distressing affection which is hard to remove. In most of these cases there are probably conditions of irritation occurring in the muscles themselves. Other tonic spasms in the muscles of the lower extremity are rare, but individual cases of isolated tonic spasm in the abductors, in the iliopsoas, in the muscles of the calves, etc., have been observed. More extensive tonic contractures of the muscles of the leg are not very rare in hysteria, especially in the hysteria of children. The so-called saltatory reflex spasm is undoubtedly also hysterical (*q. v.*).

7. SPASMS IN THE RESPIRATORY MUSCLES

Tonic spasm of the diaphragm has been seen in a few rare cases. The lower part of the thorax is much expanded, the epigastrium is protruded, and the respiration, which shows intense dyspnœa, can be performed only by the upper part of the thorax. The depression and immobility of the diaphragm can be made out on percussion. Many patients have a severe pain in the region of the diaphragm. The condition is not without danger and demands instant interference: inhalations of chloroform, subcutaneous injections of morphin, a warm bath with a cool shower bath after it, faradization of the skin in the region of the diaphragm, galvanization of the phrenics, etc.

Clonic Spasm of the Diaphragm, Singultus.—The well-known "hiccough" or "sob," due to a sudden spasmodic contraction of the diaphragm, associated with a brief inspiratory sound, is in its mild forms a very frequent condition, which soon passes off; but it sometimes increases to a persistent, obstinate, and very troublesome affection, which may last for weeks or months. Hysterical hiccough is the most frequent. This comes on especially after mental excitement, and it may sometimes last, with short interruptions, for weeks or even months. Persistent hiccough may also be excited reflexly in affections of the stomach, intestines, peritoneum, etc. In some cases the hiccough depends upon a direct lesion of the phrenic nerve, as we have seen in one case of tubercular mediastino-pericarditis. We have also seen hiccough lasting for hours in cerebral apoplexy, and in chronic myelitis extending to the cervical cord.

In the milder cases hiccough soon passes off without special treatment. Holding the breath, pressure on the closed glottis, blows on the back, etc., are procedures generally known to the laity, and they are often used to suppress hiccough. In hysterical hiccough intelligent and at the same time energetic mental treatment will often rapidly lead to the voluntary suppression of the spasmodic movements and thus to complete recovery. Such treatment is often aided by the influence (in part merely suggestive) of some internal remedy, such as bromid of potassium, or the use of electricity. In severe hiccough, due to organic disease, we must sometimes resort to narcotics (large doses of bromid of potassium, opium, morphin, inhalations of chloroform). The constant current to the phrenic nerve or faradization of the region of the diaphragm sometimes acts beneficially.

Complicated respiratory spasms, either in the form of spasmodically accelerated and forced breathing, or spasms combined with all sorts of other symptoms, with many gurgling noises, eructations, etc., are almost exclusively confined to hysteria. We have ourselves counted in one such case over two hundred respirations a minute! The best remedy for most of these forms of spasm, and one which often acts instantly, is a cool bath with energetic cold shower baths. The yawning spasm (*chasmus, oscedo*), sneezing spasm (*sternutatio convulsiva, ptarmus*), laughing and weeping spasms, coughing spasm, etc., also belong to the respiratory spasms. We once saw an instructive example of the latter in a boy ten years old, undoubtedly due to hysteria. A peculiar, hollow, barking cough came on as a "reflex" (that is, due to association of ideas) either spontaneously or on pinching any part of the skin. The affection lasted for some weeks, and then disappeared quite suddenly.

A nervous cough, often of a peculiar spastic character, is not an infrequent occurrence in hysterical and nervous people.

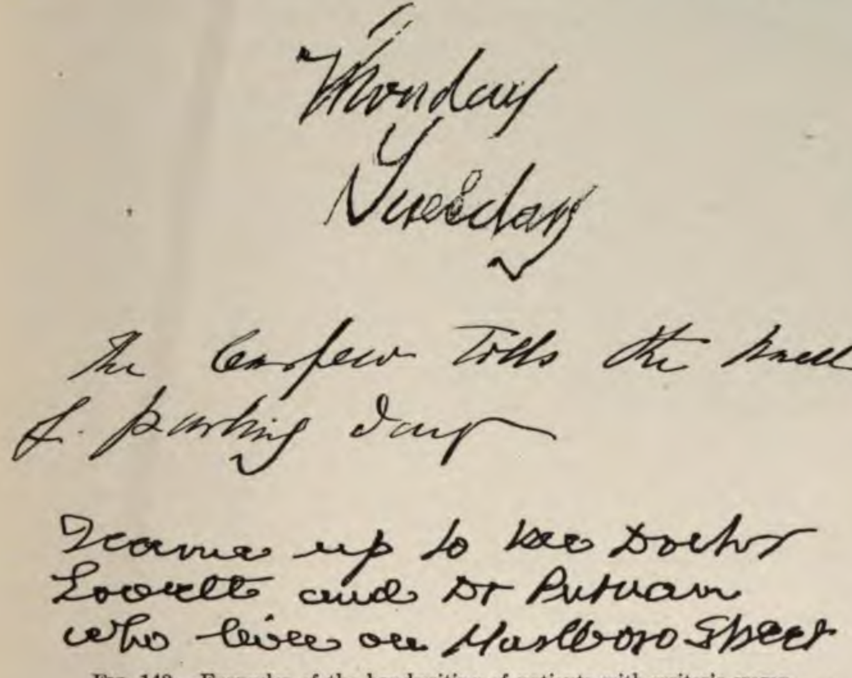
CHAPTER IV

WRITER'S CRAMP AND ALLIED PROFESSIONAL NEUROSES

Writer's cramp (*graphospasm, mogigraphia*) is the commonest form of a whole class of peculiar disturbances of motion, to which Benedikt gave the appropriate name of "professional neuroses of coördination." Their characteristic feature is that the disturbance in a certain group of muscles appears only when these muscles come into harmonious action, in some definite and usually some delicate and complicated occupation. Although the persons who suffer from writer's cramp can move and use the muscles of their right arm and hand for ordinary purposes in a perfectly normal manner, these same muscles at once refuse their service when the patient begins to write. In this case the disturbance cannot lie in the innervation of the individual muscles themselves, but it must be referred to the form of their associated action —that is, it must be a disturbance of coördination. It therefore becomes very probable that the cause of the spasm is to be sought, not in the peripheral parts, but in the centers. We are convinced that writer's cramp and the allied occupation neuroses are due to disturbances of the central (cortical) innervation. Writer's cramp is a morbid condition in writing similar to stuttering in speaking. Overexertion in writing plays the most important part as an ætiological factor. Writer's cramp is therefore seen chiefly, but of course not exclusively, in those persons whose occupation entails continuous writing, especially in secretaries, merchants, bureau officials, etc. A general nervous predisposition seems also very notably to increase the tendency to writer's cramp. Most cases are associated with pronounced neurasthenic symptoms. Mental factors (fright, anxiety) sometimes play an unmistakable part (*vide infra*). Attention has also been called to the fact that bad pens, such as hard steel pens, improper methods of holding the pen in writing, etc., may favor the development of writer's cramp.

Symptoms.—The essential symptom of writer's cramp is the appearance of certain disturbances at every attempt to write, which render writing very difficult or entirely impossible. The affection usually begins gradually, but increases quite rapidly. For the precise characterization of the disturbance Benedikt has distinguished three forms of writer's cramp, but they run into one another in various ways. The spastic form is the most frequent. The patient scarcely begins to write when contractions or tonic spasms come on in the different fingers. The pen is either pressed spasmodically deep into the paper, or, when it is moved, it makes very faulty irregular movements. We very often see a tonic spasm of the pronators of the forearm set in on each attempt to write. Writing is, under such circumstances, wholly impossible, or it is done only with the greatest exertion, and the characters are also utterly distorted, unequal, and mingled with false strokes and blots (*vide* Fig. 142). Finally, in the tremulous form of writer's cramp there is such a marked tremor in the right hand at every attempt to write that the letters are completely illegible. We have seen several such cases in children, which we have regarded as distinctly hysterical.

In the paralytic form the disturbance is chiefly a tired feeling in the right arm of rapid onset, like paralysis, and it is often associated with pain. This form of disturbance in writing ought not properly to be classed with writer's cramp, since it is absurd to speak of a "paralytic" form of "cramp."



Monday
Tuesday

The Curfew Tolls the knell
of parting day

I came up to see Doctor
Lovett and Dr Putnam
who lives on Marlboro Street

FIG. 142.—Examples of the handwriting of patients with writer's cramp. (From specimens furnished by Dr. J. J. Putnam.)

As has already been said, the motility of the right arm in every other respect is usually perfectly normal, but sometimes analogous symptoms also appear in the same patient in many other fine employments, such as sewing, piano playing, etc. The sensibility is usually unaffected, except for the muscular pains already mentioned, and a frequent feeling of numbness in the forearm and fingers. A few painful pressure points have sometimes been found on the cervical and dorsal vertebræ. We should also examine the peripheral nerves, since painful thickenings are sometimes said to be found in them (although I do not myself share this view), which possibly stand in a causal relation to the disease. If we are dealing with people who are generally neurotic, as is almost always the case, they often complain at the same time of headaches, mental uneasiness, general weakness, etc. In such cases the degree of the disturbance, as we have said, is always very much influenced by mental conditions (excitement, anxiety), just as is the case in stuttering. We knew a patient who could not write a word, in spite of the greatest effort, if any one were looking at him, but otherwise he wrote a beautiful flowing hand.

Diagnosis.—The diagnosis of writer's cramp is almost always easy. We must guard against confusing it with other nervous diseases, such as chorea, paralysis agitans, multiple sclerosis, beginning muscular atrophy, or agraphia, which, of course, under some circumstances may lead to disturbances in writing.

Prognosis.—The prognosis is always to be given with reserve. Complete recoveries without doubt do occur, but many cases are extremely obstinate and others are incurable. Relapses are also very common even after improvement has set in. Some patients are obliged to choose another calling in consequence of their trouble.

Treatment.—The treatment begins first with the command to give up writing entirely for several weeks. If this command can be obeyed, the mere rest may be of service in mild incipient cases. Certain contrivances for writing, which the patient can best test himself, are often of advantage, such as sticking the penholder through a cork, using a large penholder, a change in the way of holding the pen and in the position of the arm, etc. Nussbaum has invented a kind of bracelet to which the penholder is fastened, and which is held firmly by the outspread fingers. Learning to write with the left hand, which is often tried by patients, does not always lead to a good result, since the cramp occasionally appears in the left hand also.

Of the special methods of treating writer's cramp the application of galvanism deserves the first mention. Avoiding all strong currents and variations of the current, we apply the stabile anode to the brachial plexus, and also to the different nerves (especially if they be sensitive to pressure) and the affected muscles, for five or ten minutes. The cathode is placed in the vicinity of the cervical vertebræ. If painful points are found they receive special treatment. We may also use galvanism, as an experiment, through the head. Of late massage, and especially methodical gymnastics, have shown still more favorable results than electrical treatment, but the use of them demands special technical skill, and therefore they have so far obtained excellent results chiefly in the hands of certain specialists; but such a methodical reëducation in the movements employed in writing should always be tried. We commence with simple writing movements executed slowly and in rhythm, and then progress gradually to more difficult letters. We can very rarely promise success from internal remedies, such as subcutaneous injections of strychnin, atropin, etc., but those methods of treatment often act favorably which aid the general strength of the nervous system, such as cold-water cures, sea bathing, and residence at a mountain resort. In writer's cramp, just as in stuttering, we see very plainly the influence of mental factors (rest, increasing or diminishing self-confidence, excitement, etc.) upon the trouble.

As an appendix we will mention here some other professional neuroses that are occasionally seen. They are piano-player's cramp, which is seen especially in young conservatory pupils, violin- and 'cello-player's cramp, telegrapher's cramp, tailor's cramp, milker's cramp, the peculiar disturbances of innervation in the hands which often occur in cigar rollers, etc. In the lower extremities there seems to be an analogous affection in ballet dancers, and also in sewing-machine girls, turners, etc. We have seen professional cramp in the tongue in a clarionet-player. The special points in the symptomatology and treatment of all these forms of cramp are in large part analogous to the conditions described in writer's cramp. In piano-players the neurosis appears chiefly in the paretic form—mild fatigue—and is usually associated with quite severe pain, that comes on during playing, in certain parts of the arms.

In treatment, the best results are obtained by local cold douches and massage, but general treatment (iron, cold baths, country residence) is never to be neglected. If there has been previous overexertion in playing, it must be stopped entirely. Generally it has proved better, in my experience, to continue with brief methodical exercises. Finally, it may here be noted that in certain laborious occupations that are persistently practiced a severe group of nervous symptoms may also arise. For example, Hirt has described an affection which occurs in sewing-machine girls, and is characterized by disturbances of sensibility, pain, paræsthesia, and in some cases anæsthesia, ataxia, absence of the tendon reflexes, and swaying with the eyes shut. The disease thus recalls very closely the picture of locomotor ataxia, but it is curable with proper treatment. Therefore Hirt suspected an affection of the peripheral nerves. Similar symptoms are also seen in other classes of laborers. Thus, for example, I have repeatedly seen peculiar spastic paretic conditions of the legs after continuous fatiguing work in the fields in a stooping position (digging carrots, etc.).

CHAPTER V

SIMPLE AND MULTIPLE DEGENERATIVE NEURITIS

I. SIMPLE NEURITIS

1. **Primary Simple Neuritis.**—In the previous chapters we have become acquainted with a series of morbid conditions which most probably are to be referred to primary inflammatory changes of a particular peripheral nerve. We have seen that many cases of sciatica and of neuralgia in other nerve regions, and probably, too, all the so-called rheumatic peripheral paralyses (rheumatic facial paralysis, deltoid paralysis, etc.), are due to such a neuritis. We have very little information as to the special causes of such forms of neuritis; we may only suspect that in such cases there are definite infectious or toxic substances which act on the affected peripheral nerves. In many cases, especially in most neuralgic affections, it seems rather to be slight inflammatory changes in the nerve sheath and the interstitial connective tissue of the nerve, but in all forms of neuritis leading to motor paralysis it is chiefly a destruction of the nerve fiber itself caused by the action of the injurious substance ("parenchymatous inflammation," or, better, "degenerative neuritis"). In severe, and especially in acute, cases the parenchymatous and the interstitial changes may develop simultaneously. We will go more fully into the description of the anatomical details in the section on "multiple neuritis" below.

We may also mention here, as of practical importance, those cases of primary simple neuritis which, after the analogy of facial paralysis which has been fully described, lead to acute peripheral paralysis in other nerve regions. To this class belong, first of all, neuritic axillary paralysis (deltoid), neuritic paralysis in the ulnar, long thoracic (serratus anticus major), crural, peroneal, etc.; neuritic plexus paralyses also occur, especially in the brachial plexus. In many, although, of course, not in all of these cases, an exposure to cold is to be looked upon as the cause of the neuritis ("rheumatic neuritis"). The disease itself usually begins with severe pain of greater or less intensity and paræsthesia in the neighborhood of the affected nerve. This symptom, which possesses the greatest diagnostic significance, especially in affections of the motor nerves, is generally attributed to an involvement of the nerve sheath and interstitial tissue (*nervi nervorum!*). At the same time with the pain, or soon after it, appear signs of motor weakness in the muscles supplied by the affected nerve. The paralysis may vary extremely in intensity. In mild cases the symptoms soon disappear and recovery ensues. In severer forms electrical reaction of degeneration and atrophy of the paralyzed muscles develop; but even here, as has already been said, we may usually expect a favorable issue. The affected nerves are often very sensitive to pressure. Slight disturbances of cutaneous sensibility are often to be made out on careful examination when there is neuritis of a mixed nerve trunk; but, as a rule, the motor disturbance is almost always much more apparent than the sensory. The treatment follows the methods in vogue in all peripheral paralyses.

2. **Secondary Simple Neuritis.**—There can scarcely be a doubt that secondary neuritis may occur in consequence of disease of neighboring organs. Neuritis sometimes develops in open wounds which permit the direct entrance of inflammatory agents to the nerve; the inflammation is distributed chiefly in the sheath and the connective tissue of the nerve. It is, however, of rare occurrence—much rarer than we were formerly disposed to assume. Simple mechanical injuries and lacerations of peripheral nerves are much commoner, occurring in all sorts of affections in their neighborhood—fractures, luxations, caries of the bone, tumors, etc. In such cases we should not speak, without qualification, of "inflammation," since we usually have to do, as has been said, with purely mechanical consequences. Apart from the ordinary secondary descending degeneration (*vide supra,* page 199), such mechanical lesions of the nerves have in themselves no tendency to extend upward in the form of a "*neuritis ascendens,*" or "*migrans,*" as was formerly believed, and the assumption of an "ascending neuritis," following diseases of internal organs (sexual organs, kidneys, intestines, etc.), and finally leading not infrequently to secondary myelitis, so-called "reflex paralysis," etc., is warranted only in the rarest cases. It is certain, however, that inflammatory processes may sometimes attack neighboring nerve trunks, and that an acute inflammation may sometimes extend immediately up the neurilemma of the outgoing nerve to the cerebral and spinal meninges. So far as these processes deserve clinical significance they will receive special mention in the appropriate places.

[It has long been known that, when a nerve has been cut, the portion below the injury rapidly degenerates—Waller's law of degeneration. The portions that degenerate are the axis-cylinder processes that have been separated from the cell body of the neuron. As a result of more delicate methods of research, it has been found that the rest of the neuron—the cell body and the axis-cylinder stump—also undergoes slighter changes. There may be, therefore, really an ascending degeneration in the nerve, but it is very slight and has little or no clinical significance.—K.]

II. MULTIPLE DEGENERATIVE NEURITIS

Ætiology.—Although the occurrence of extensive primary disease in the peripheral nerves was practically unknown not very long ago, many investigations in the last few decades (Dumenil, Joffroy, Leyden, and many others) have taught us that no part of the entire nervous system is so much exposed to all possible harmful influences as the peripheral nerve fiber. We are at present acquainted with a long list of types of nervous disease which are due exclusively, or at least chiefly, to more or less extensive degeneration of the peripheral nerves. The more we investigate the causes of these diseases, the more we become convinced that it is almost always, in the last instance, chemical poisons which enter the body or are formed in the body itself and exert a destructive influence on the peripheral nerve fiber. It is indeed possible that all such poisons act from the outset upon the whole peripheral neuron—that is, the ganglion cell and peripheral nerve fiber—but the visible injury (degeneration) is generally manifested exclusively in the peripheral nerve fiber, and probably first in the branches most remote from the trophic center, the nerve cell. Only when the action of the poison is severe and long-continued can the whole neuron—that is, the motor ganglion cell in the anterior horn of the cord—be visibly injured or even completely degenerated. [Changes may also occur in the peripheral sensory neurons, not only in the peripheral portions of their nerve fibers, but also in their ganglion cells in the spinal ganglia and in their axis-cylinder processes in the spinal cord. —K.] The toxic origin of peripheral-nerve degeneration is most clearly seen in those morbid conditions which we can refer with certainty to a definite, well-recognized poison. These are the special "toxic neuritides," in the strict sense of the word, with which we have already become acquainted in the description of lead and arsenical paralyses. We have seen that, by the action of these poisons, extensive destruction of the peripheral nerves may ensue, which, of course, is followed by serious clinical symptoms. Disregarding several other rare forms of poisoning (copper, zinc, silver, phosphorous, mercury, bisulphid of carbon, etc.), there still remains one extremely important and comparatively common form of poisoning which leads to multiple neuritis, and that is chronic alcoholism. Alcoholic neuritis is by far the commonest form of multiple neuritis, and it will therefore require a special description later.

Besides these neuritides, directly of toxic origin in the strict sense of the word, we find a second large group of cases which come on secondarily in the course of, or as a sequel to, other diseases. To this class belong, in the first place, most of the cases already mentioned (page 197) of "paralysis after acute disease." Since it is usually an acute infectious disease (diphtheria, typhoid fever, scarlet fever, septic infection, puerperal disease [pneumonia], etc.), we may reasonably assume that it is the toxins formed under the influence of the corresponding agents of infection which lead to the death of the peripheral nerve fiber; but similar conditions also occur in chronic infectious diseases. Tuberculosis in particular seems to stand in close relation to multiple neuritis, although, of course, it is often hard to exclude complication with some septic infection or the simultaneous action of other intoxications (especially alcohol, *vide infra*). [A more or less extensive neuritis is not uncommon in leprosy. The distribution of the neuritis is less symmetrical than in other forms of neuritis, and the morbid changes in the peripheral nerves, which involve the perineurium and interstitial tissue more than the nerve fiber itself, are thought to be due to the direct invasion of the nerves by the bacteria rather than to the general toxic effect of the disease. The chief symptoms are marked anæsthesia and muscular wasting. The anæsthesia in the anæsthetic form of leprosy is due to these lesions of the peripheral nerves.—K.] It is certain that metasyphilitic nervous diseases (tabes, *q. v.*) are due, at least in part, to degenerations of the peripheral nerves; but in these cases there is usually extensive disease at the same time in the central nervous system, so that we do not class these conditions as true neuritis. [Syphilitic processes may, of course, involve individual nerves, somewhat in analogy with leprous neuritis, but true multiple neuritis may rarely be of syphilitic origin. Malaria is also said to cause multiple neuritis. Neuritis is more apt to follow the pernicious forms of malaria occurring in the tropics—Jamaica, India, Madagascar, etc. It may come on suddenly during the comatose period of a pernicious attack. In some of the acute cases there have been well-marked changes in the nerve sheaths, in other cases there have been ordinary degenerative changes. The neuritis has been referred to the toxins produced by the plasmodium. Trophic and vasomotor symptoms (cyanosis) seem more marked than in other forms of neuritis. There is a possible relation between malarial neuritis and beri-beri (*q. v.*).—K.] Of noninfectious diseases, diabetes mellitus, first of all, is sometimes most closely related to the onset of multiple neuritis. Here, again, we can scarcely help thinking that the nerve degeneration has been produced by some abnormal chemical product, but the question of diabetic neuritis is still in great need of further investigation. It is a fact that neuritic affections of various sorts occur in severe and also in mild forms of diabetes, but of course we may often think, on the other hand, that the glycosuria is only a sequel of the neuritis, or that it has come on at the same time with it (from the same cause, especially previous alcoholism). In a few cases polyneuritis has been seen in severe (usually ulcerative) carcinomata, but in these cases secondary septic processes may play the chief part, as in tuberculosis (*vide supra*).

The third group, finally, is composed of those cases of multiple neuritis which must be termed primary multiple degenerative neuritis. Of course, alcoholic or diabetic neuritis, for example, may come on apparently as a primary disease, but the true primary form of the disease includes only those cases where no other special preceding cause can be discovered, and where the neuritis apparently comes on as an independent disease, due to a special specific cause. The whole course of the disease, which often has an acute febrile character (*vide infra*), shows that we must in such cases also consider toxic-infectious influences, but we know nothing as yet as to the precise form of this infection. We can sometimes find no special cause. There has often been previous exposure to severe cold. Such cases are called " rheumatic multiple neuritis," without, of course, implying that they have any definite connection with articular rheumatism.

In other cases, on careful investigation, we may find some relation between the neuritis and a preceding slight infectious or toxic condition (tonsillitis, intestinal disorders, etc.). At times, however, no special cause for the polyneuritis can be determined. It is worthy of mention that primary polyneuritis has been described by several physicians as occurring in epidemic form. [Epidemic polyneuritis may have some relation to epidemic poliomyelitis (*q. v.*). The peculiar form of epidemic neuritis known as beri-beri will be spoken of later.—K.]

We must add in conclusion that we have very often seen cases of multiple neuritis where we could discover several of the above-mentioned causes at the same time, especially when alcoholism and tuberculosis or alcoholism and diabetes mellitus could unite their noxious effects. We have also seen alcoholic polyneuritis come on directly after some acute febrile disease (pneumonia, etc.), which distinctly reminds us of the appearance of delirium tremens in acute diseases.

Pathological Anatomy.—The anatomical changes of neuritis consist, as has been said, mainly in a degenerative destruction of the nerve fibers. We do not know whether the toxic agents act directly on the peripheral nerve fibers or on the peripheral motor neurons in their entirety. The visible coarse anatomical destruction is usually found, at all events, only in the peripheral offshoots of the neuron—that is, in the "peripheral nerves"—and apparently it is most marked in the most distant terminal arborizations of the nerves. In the larger nerve trunks and plexuses the changes are usually much less pronounced than in the smaller nerve branches.

Only in a few acute cases do the interstitial connective tissue and the nerve sheath show the changes of true acute inflammation. In such cases we find the affected nerves swollen and decidedly reddened from the great vascular injection; with the naked eye we can sometimes see several or many small hemorrhages. The microscope shows the nerve sheath and the interstitial tissue infiltrated with many round cells, but, as a rule, the interstitial tissue is little or not at all involved in the disease. The pathological anatomical process consists exclusively of a destruction of the axis cylinder and medullary sheath (see Fig. 143). If we stain the diseased nerve fibers with osmic acid we see the sheath of Schwann filled with many large and small flakes and globules of the medullary substance of the nerve which are stained black. The fatty globules formed by this degeneration are taken up by individual leucocytes (perhaps also by the endothelial cells?), and thus the so-called "fatty granular cells" are formed. If the process has lasted some time and the acute symptoms have ceased, either the regeneration of the nerve fiber begins, or, when the special nerve tissue is permanently destroyed, there is a new formation of an abundant interstitial connective tissue. If we now examine a transverse section of a diseased nerve we find the individual nerve fibers no longer close together, as under normal conditions, but separated from one another in many places, where some fibers have wholly disappeared, by a large amount of interstitial connective tissue. If the new formation of connective tissue attains a higher degree, the nerve then becomes firmer and denser than normal, and, when there is a very large amount of connective tissue (after the analogy of callus formation), it also becomes thicker, so that in some places we may sometimes speak of a neuritis nodosa. If, however, the polyneuritis recovers, it is due to regeneration—that is, to a complete new growth of the destroyed nerve fiber. The remarkably great power of regeneration of the peripheral nerves is one of the most important facts in the pathology of the nervous system. It is explained in a simple manner by the persistence of the cell of origin (the motor ganglion cell in the anterior horn) from which the destroyed terminal branches grow out again like the severed branches of a tree. Therefore the severest peripheral paralyses and ataxias may sometimes completely recover.

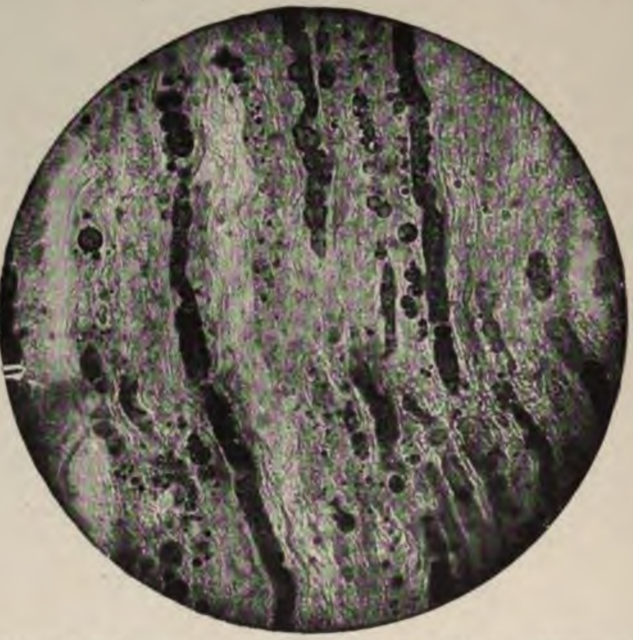


FIG. 143.—Radial nerve in alcoholic polyneuritis. Osmic-acid preparation. (Personal observation.)

In the cases of "chronic multiple neuritis" the disease succeeds an acute neuritis or develops in an insidious fashion from the start. In the latter case the first acute stage of hyperæmia and cell infiltration is wholly wanting and the disappearance of nerve fibers and the secondary new growth of connective tissue is a chronic process from the first. Such conditions perhaps should not properly be called "neuritis," but "primary chronic degenerative atrophy of the nerves"; but the name is of less consequence than a clear idea of the nature and development of the morbid process. There is also nothing strange in finding in some cases slight changes in the motor ganglion cells themselves. When the action of the toxic substance is severe or long continued the whole motor neuron, including its cell of origin, suffers. The paralyzed muscles in every peripheral neuritis soon become very much atrophied. Microscopic examination shows all stages of atrophy of the fibers up to complete disappearance. The transverse striation is, for the most part, preserved, and the number of the muscular nuclei is much increased. In individual cases we also find degenerative destruction of the muscular fibers, hypertrophy, swelling, waxy degeneration, etc. A genuine myositis—that is, the formation of small inflammatory foci in the interstitial tissue—is sometimes joined to the neuritis. In such cases of "neuromyositis" the poison seems to act from the start simultaneously on the nerves and muscles.

Besides its action on the peripheral motor neurons and the muscles, the action of the morbific influence may extend still further. Thus we find coexisting changes in the columns of Goll, and even in the brain, which excite special clinical symptoms.

Symptoms and Course of the Different Forms of Multiple Neuritis.—In order to attain a correct understanding of the symptomatology of multiple neuritis, we must first remember that the disease is not ætiologically one absolutely single affection, and that therefore we must expect *a priori* certain differences in the clinical picture. These differences are due not only to the degeneration of different nerve trunks, but also, in part, to the fact that the same specific variety of nerve fiber is not always the one most affected. In general, of course, the rule undoubtedly holds good that the motor nerve fibers afford the least resistance to all the injurious influences previously mentioned. Peripheral paralyses, then, are the chief and most common symptom of polyneuritis. The peripheral sensory neurons, however, are not always spared by toxic agents. Primarily, the involvement of the sensory nerves is shown by symptoms of nerve irritation (pain, paræsthesia). It is, of course, difficult to decide whether these neuritic pains are produced by direct irritation, or by interstitial inflammatory changes. On careful examination, however, sensory disturbances (anæsthesias) are found frequently enough, and particularly, as we will learn later on, in the distribution of the nerves coming from the deeper structures (muscles, etc.). Occasionally, even, these nerves are almost alone the seat of degenerative changes, and then a true ataxia (compare page 204) is necessarily produced. There may be some doubt, however, in such cases (though this is of no importance as regards the result), whether the degeneration is limited entirely to the peripheral dendrites of the sensory neurons, or whether it also affects the spinal prolongations into the posterior columns. At any rate, from a symptomatological standpoint we must distinguish two forms of neuritis—a paralytic form (the usual form) and an ataxic form (so-called acute curable ataxia, and also, especially, the pseudo-tabes of alcoholic subjects); but combinations of the two forms occur, since paralytic conditions and disturbances of coördination may be united.

Passing now to the description of the general aspect and course of polyneuritis, it seems best to keep separate the different forms which may be distinguished either clinically or ætiologically.

1. Primary Acute and Chronic Multiple Neuritis

The disease usually begins acutely, sometimes almost in an apoplectiform manner, and without any definite occasion, precisely like an acute infectious disease. Febrile symptoms, with temperatures from 102° to 104° F. (39° to 40° C.), come on in persons previously in good health, usually in adults before or during middle life, with severe general disturbance, loss of appetite, dullness, headache, and sometimes even mild delirium. In these acute cases albuminuria and a slight enlargement of the spleen have sometimes been observed, which symptoms also point toward the infectious nature of the disease. In other cases the initial general symptoms are much less severe, or they may be entirely wanting. The nervous symptoms then occur, apparently without any cause and without any other pronounced disturbances, in a fairly acute manner, in previously healthy persons. The pains, which are hardly ever absent, are very characteristic, and will generally lead the experienced physician at once to the correct diagnosis. They are described as pulling and tearing, are felt chiefly in the loins and the extremities, and sometimes follow approximately the course of the large nerve trunks. On passive motion and pressure on the deeper structures, the pains become particularly pronounced. Since in some cases a number of the joints are swollen, the disease at first may be mistaken for acute articular rheumatism. The first symptoms of paralysis, usually in the lower extremities, appear very soon after these initial symptoms, or almost at the same time with them. The patient notices that he cannot readily move one leg, and soon after he notices the same of the other. The paralysis may remain confined to the legs, but more frequently it extends to one or both arms. In regard to its distribution, the forearms and lower legs are generally more affected than the upper arms and thighs, and the extensors (radial and peroneal region) are also more affected than the flexors; but other varieties of distribution may occur—for instance, affection of the glutæi and thigh muscles, etc. The pronounced symmetry of the paralyses in the two halves of the body is very noticeable and characteristic of polyneuritic paralysis, in contradistinction to poliomyelitic paralysis.

If we examine the paralyzed parts more carefully we find a perfectly atonic and more or less extensive paralysis. The reflexes are almost always diminished, the tendon reflexes are usually entirely absent, and the cutaneous reflexes are weak, or they also have almost wholly disappeared. In only a few cases are the reflexes increased—a symptom which is to be regarded as analogous to cutaneous hyperæsthesia. We can usually make out, after a few days, a decided diminution of electrical excitability in the affected nerves and muscles, which soon becomes a pronounced reaction of degeneration. If the paralysis is of longer duration, there is a decided atrophy of the muscles. In these cases the severe initial symptoms of sensory irritation, as a rule, rapidly disappear, although slight pains, paræsthesia, and especially a considerable sensitiveness of the paralyzed parts to pressure and to passive motion, often last for a long time. In many acute cases the hyperæsthesia of the skin and of the deeper parts reaches a very high degree. Objectively, the disturbances of sensibility are very slight in many cases, although on careful testing we can frequently enough find disturbances of sensibility, particularly in the distal ends of the extremities. It is only necessary to test carefully all forms of sensation, especially sensibility to deep pressure and muscular sensibility. Other observers and we ourselves have also repeatedly seen the symptom of so-called delayed conduction—that is, delayed sensation of pain (*vide supra,* page 151). We usually find no disturbances in the distribution of the cerebral and bulbar nerves. An affection of the optic nerve has been mentioned in only a few cases. Facial paralysis and, much less frequently, paralysis of the tongue or of the ocular muscles may also occur. We have ourselves repeatedly seen total bilateral facial paralysis. We may mention as a rare complication acute mental disturbances (confusion, anxiety, weakness of memory, *vide infra*). Details of this very peculiar "polyneuritic psychosis" will be given below in the description of alcoholic polyneuritis. The marked increase in the frequency of the pulse, which is usually present, is important, and probably depends upon a disturbance of the vagus. Trophic disturbances in the skin, hair, and nails are not very rare. Profuse sweating sometimes occurs. Œdematous swelling of the affected extremities has also been repeatedly observed. The functions of the bladder and rectum, however, almost always remain unaltered, but in some cases slight disturbances of the bladder may, of course, be discovered.

In regard to the course of the disease, in the severest cases it may soon terminate fatally, almost always because the paralysis extends to the muscles of respiration. The inspirations are labored, and are performed with the upper part of the thorax only, while the epigastrium is motionless, or is retracted on inspiration from the paralysis of the diaphragm. There is also paralysis of the other muscles of respiration, the abdominal muscles, etc., so that, after the disease has lasted a week or ten days, death ensues with all the signs of respiratory insufficiency. Such cases have been repeatedly described in the past under the title of acute ascending paralysis (Landry's paralysis, *q. v.*). A second class of cases also begins quite acutely, but then takes a chronic course. The initial acute febrile symptoms cease after a few days, although the paralysis continues to develop to some extent. Then the affection seems to come to a standstill, and the first signs of improvement gradually begin to appear. Since there is a more or less pronounced atrophy of the muscles in these cases, it always takes considerable time—usually several months—for recovery; but it is often astonishing to see how the most extensive paralysis may finally get entirely well, even after six months' or a year's duration. A third class of cases follows a chronic course from the outset, although even in these cases there may be more acute exacerbations of the disease. In these cases quite extensive atrophic paralysis gradually develops in the lower extremities, and usually in the upper extremities also. The reflexes disappear; the sensibility is, as a rule, somewhat, but very rarely, much diminished. Pain is always present at first, but later on in the disease it often becomes subordinate. The bladder and rectum usually remain normal in their functions or show only slight disturbances. If the disease advance gradually it may terminate fatally even at a late period, after a course of months, usually from a final paralysis of respiration; but, on the other hand, even after a protracted course, the disease may come to a standstill, and even progress to a complete, or at least to a partial, recovery.

The *diagnosis* of multiple neuritis is, as a rule, easy for one who is acquainted with the disease and notes the different symptoms carefully. In regard to diagnosis, the chief importance should be placed on the generally acute beginning, with pronounced symptoms of sensory irritation, with frequently a very considerable sensitiveness of the nerves and muscles to pressure and general cutaneous hyperæsthesia; and also on the appearance of a rapidly extending paralysis, whose peripheral nature may be attested by the presence of reaction of degeneration, muscular atrophy, and the absence of the cutaneous and tendon reflexes. Such a paralysis can be produced by nothing but an affection of the peripheral nerves or poliomyelitis (*vide infra*). This latter disease was, in fact, formerly confused with multiple neuritis, but a careful attention to the initial symptoms, especially to the pain and disturbances of sensation, usually makes the differential diagnosis possible.

The *prognosis* of multiple neuritis is doubtful, as is shown by the description of the course of the disease, but it is by no means very unfavorable. If the patients get through the first acute stage of the disease without accident, we may hope for recovery, or at least actual improvement, even with extensive paralysis. Such striking results in the way of recovery, after paralysis that has lasted for months, are also important in regard to diagnosis, since such extensive processes of regeneration are possible in affections of the peripheral nerves, but scarcely in spinal diseases with destruction of the motor ganglion cells, and hence they are sometimes an additional confirmation of the diagnosis of neuritis.

Treatment.—In the first stage of the disease, especially if severe pains, swelling of the joints, or high fever be present, it is advisable to try the exhibition of salicylic acid, from which several observers have seen a favorable effect. We give 10 gr. (gm. 0.5) of the acid every hour, or a few larger doses of salicylate of sodium, 1 drachm to 1½ drachms (gm. 4 to 6). Instead of salicylic acid we have used antipyrin and phenacetin, salipyrin, aspirin, and similar remedies, with good results. When the pain is very severe we must use narcotics, such as injections of morphin. Embrocations of chloroform, and sometimes protracted warm baths and careful sweating, have also a palliative effect. In the further course of the disease proper care, a suitable position for the limbs, and diet—nourishing food—are the main things for the patient. The regenerative processes of recovery begin spontaneously, if they begin at all, but we may hasten recovery and make it complete by a subsequent electrical treatment, especially galvanism. For the completion of the recovery, bathing (simple warm baths, salt baths, etc.) is serviceable, and also the baths at Teplitz, Wiesbaden, Nauheim, and Oeynhausen, and the cautious use of massage. Of the internal remedies, the so-called tonics (preparations of iron, quinin, strychnin, arsenic, etc.) may be used.

2. The Ataxic Form of Multiple Neuritis

(Acute Curable Ataxia)

We cannot make a sharp division between the ataxic and the paralytic forms of multiple neuritis. Both conditions, paralysis and ataxia, may occur in the same patient. It is important, however, to know that almost pure ataxia (without paralysis) may develop acutely, and that it is undoubtedly to be referred to degeneration of the peripheral, centripetal sensory neurons either in their peripheral or spinal branches.

Under the ataxic form of polyneuritis must be classed, first, the cases of ataxia repeatedly observed after various acute infectious diseases, especially ataxia after diphtheria (*q. v.*), and also after typhoid fever, smallpox, septic affections, dysentery, etc.; but there is also a primary acute ataxia, which comes on without any discoverable cause or after exposure to severe cold in previously healthy persons. Severe general initial symptoms are, as a rule, not present, and the pain is not very great. The ataxia usually develops in the legs, more rarely in the arms. There may be slight disturbances of sensibility at the same time, but not necessarily. The patellar reflex is usually, but not always, absent. As a rule, with proper care and nursing the patient recovers completely in a few weeks. The diagnosis of this primary acute ataxic form of polyneuritis is usually not difficult. The disturbance of the patient's gait may at first arouse the suspicion of tabes, but the rapid onset, the perfectly normal pupillary reflexes, and usually also the normal micturition, oppose it. We often find in some muscular regions a true paresis besides the ataxia, which is not like the conditions in tabes. We once saw, for instance, a severe ataxia of the legs associated with double facial paralysis. Paralysis of the legs and ataxia of the arms may occur. In any doubtful case a favorable termination in recovery forms the final criterion in favor of polyneuritis.

3. The Acute and Chronic Neuritis of Alcoholic Subjects—Alcoholic Paralysis

(Pseudo-tabes [Ataxia] of Alcoholic Subjects)

It has long been known that peculiar nervous affections often occur in alcoholic subjects (M. Huss, Leudet, and others); but formerly a disease of the spinal cord was assumed to be the cause of the symptoms, and only of late have we obtained the knowledge that the greatest part of the cases of this class are to be classed as acute and chronic multiple neuritis (Lancereaux, Moeli, and others). The practical importance of this alcoholic neuritis is not slight; first, because it may easily be confused with other nervous diseases, especially with myelitis and tabes, and, second, because its proper and timely diagnosis is of great significance in regard to treatment. Alcoholic neuritis, furthermore, is by no means rare, being certainly by far the commonest of all forms of polyneuritis. The more we consider the significance of this factor the more all other causes are thrown into the background. Not infrequently, of course, other causes must be considered at the same time with alcoholism; we see, for example, polyneuritis very often in alcoholic subjects who are also tuberculous, and sometimes the combination of alcoholism and diabetes, etc.; but in all such cases chronic alcohol poisoning probably plays the chief part. Alcoholic neuritis occurs in drinkers of spirits, and also, as we have often noticed, in habitual and excessive drinkers of beer. It is alleged to be less frequent in drinkers of wine.

Alcoholic polyneuritis, like the other varieties of multiple neuritis, occurs in different forms. According to its course we may distinguish acute and chronic cases, and according to its symptoms a paralytic and an ataxic form, but there are no strict boundaries between these forms.

Alcoholic paralysis begins quite suddenly or more gradually. Motor weakness develops, having its chief seat usually in the legs, and thus rendering walking almost wholly impossible. Frequently the paralysis involves principally the muscles of both lower legs, the dorsal extensors being affected even more than the plantar flexors of the foot. The muscles of the thigh and hip in these cases are entirely or nearly spared by the paralysis. On the other hand, we have repeatedly seen cases in which the thigh muscles (crural and gluteal distribution) are particularly affected. Such patients can occasionally still walk with effort on level ground, but they cannot go up a step or rise from a chair. With greater paralysis the patient is absolutely bedridden. Neuritic palsies are naturally of the flaccid type, with diminished muscular tonus. The paralyzed muscles atrophy rapidly, the electrical excitability diminishes, and in all fully developed cases the reaction of degeneration develops, either as a complete or as the so-called middle form of reaction (*vide supra,* page 220). The patellar reflexes and the other tendon reflexes are generally completely lost. Only a few remarkable exceptions to this rule occur.

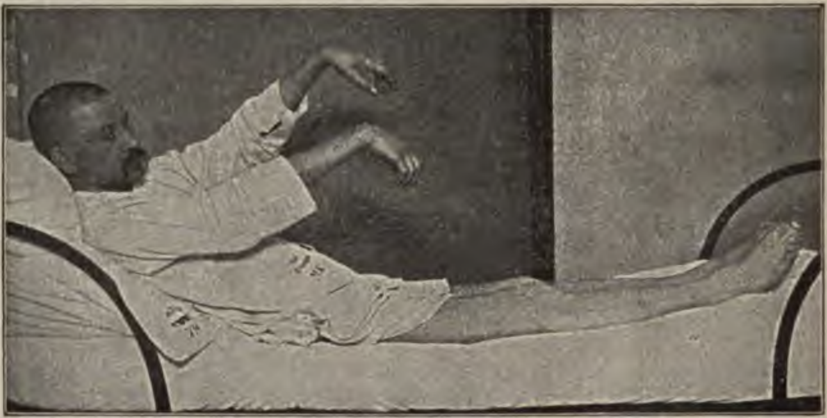


FIG. 144.—Alcoholic polyneuritis with bilateral radial and peroneal paralysis. (Erlangen Medical Clinic.)

The paralyzed muscles can also no longer be stimulated reflexly to contract by means of cutaneous stimuli. On careful testing the sensibility of the legs is rarely found to be entirely normal. In my experience we find in severe alcoholic neuritis primarily a diminution, or even a marked disturbance, of the sensations to touch, the muscular sensibility (sensations of motion and position), and the pressure sense; in other words, an interference with the conduction of the different varieties of sensory stimuli through the white posterior columns. Temperature and pain stimuli, however, are clearly perceived. For pain stimuli there is often even a pronounced hyperæsthesia, though, on the other hand, marked delay in pain sensation is not infrequently present at the same time. Often a marked œdema of the paralyzed legs develops.

In many cases the paralysis extends to the arms also, and chiefly in the radial distribution, so that both hands hang lax in volar flexion, and they and the fingers can be extended but little. The combination of paralysis of the lower leg and forearm, or of double radial and peroneal paralysis, has been often mentioned as characteristic of alcoholic paralysis (*vide* Fig. 144). Even in patients who can still extend their hands and fingers well, we often find that the strength of the movement is diminished. On the other hand, we find a combination of paralysis of the thigh muscles, with paralysis of the muscles of the upper arm and shoulder. Muscular atrophy, electrical reactions, sensibility, and the reflexes are the same as in the legs. Fibrillary contractions of the atrophic muscles do not, as a rule, occur in neuritic palsies.

Involvement of the facial muscles (facial nerve) has been observed in some cases. In some very acute and severe cases of polyneuritis there is also a paralysis of the ocular muscles, so that the eye may become completely immovable, but in the cases that have come to autopsy there seems usually to have been no neuritis of the oculomotor nerves themselves, but many small hemorrhages in the neighborhood of the third ventricle—that is, in the region of the oculomotor nuclei (so-called polioencephalitis hemorrhagica superior, *q. v.*).

Mental disturbances ("polyneuritic psychoses") have been frequently observed in alcoholic polyneuritis. Especially during the acute onset, if closely watched for, they will rarely be found to be entirely absent. They often differ little from delirium tremens, but in other cases a very peculiar type of disease develops (so-called Korsakoff's psychosis). The patient is confused, mixes up persons and places, is very restless, especially at night, apparently has hallucinations, and is consequently excited and anxious, but in the intervals is happy and contented. The most striking disturbance is an extraordinary weakness of memory for all recent events. Half an hour after eating the patient no longer knows that he has eaten. If we show him an object or utter a word or a short sentence to him, he forgets it completely in a few minutes. The more carefully we test the patient in regard to this, the more frequently shall we discover this remarkable disturbance of memory; but the recollection of the distant past and of things learned before the trouble is not at all or very slightly affected. The patient's initial confusion is chiefly the direct result of this actual failure of memory. The severe mental disturbances sometimes last only a few days, but the disturbances of memory may often be noticed for weeks, when they gradually disappear. [See the chapter on Confusional Insanity.]

The bladder and rectum are generally also unaffected by the alcoholic polyneuritis, or, at most, only slightly. [Patients with pronounced mental symptoms, however, will often pass urine and fæces in bed on account of their mental stupor.—K.] Slight elevations of temperature are not infrequent during the acute initial stage of the disease. The continued high frequency of the pulse (100 to 120), which persists for a long time even with normal temperature, is remarkable.

The ataxic form of alcoholic polyneuritis may also come on acutely, so that the patient's sudden inability to walk is due to a genuine ataxia of the legs. The ataxia, of course, is generally associated with manifest paralysis, at least in some muscular territories (glutæi, peronei, radials). The ataxia may often be masked by a greater paralysis, so that we can discover it only by careful examination. The patellar reflex is usually, but not invariably, lost; the sensibility, particularly of the deeper structures (muscular and pressure sense, *vide supra*), is found by careful examination to be almost invariably disturbed. The pains, paræsthesiæ, and sensibility to pressure of the muscles and nerves are the same as in the other varieties of neuritis. The functions of the bladder remain wholly, or almost wholly, normal.

The chronic form of alcoholic ataxia, the special pseudo-tabes of alcoholic subjects, is especially important because it is often harder to diagnosticate. In this form a type of disease develops comparatively slowly, which may closely resemble genuine tabes: ataxic gait, lancinating pains, sometimes even a sort of girdle sensation, absence of patellar reflex, slight disturbance of the bladder, definite disturbances of sensibility, etc. The disease resembles true tabes still more closely if temporary double vision occurs. In such cases we can avoid mistaking it for true tabes only by careful examination. The most important mark of distinction is afforded by the pupils, which almost always preserve their normal reaction in pseudo-tabes. We may also note that in pseudo-tabes the disturbances of the bladder are usually slight, and they may be entirely absent; that the muscles and nerves are tender on direct pressure, and that from the outset we can usually find besides the ataxia a slight genuine paresis, which in some muscles (*vide supra*) may be marked. [We rarely if ever find in pseudo-tabes the anæsthesia or analgesia over the lower part of the chest, on one or both sides, in the area of the third to the seventh dorsal roots, so characteristic of true tabes.—K.] Finally, of course, the causal factor, pronounced chronic alcoholism, is an essential consideration. It is worthy of note that in alcoholic pseudo-tabes the anatomical lesion is probably not often limited to the peripheral nerves, but also involves the spinal processes of the spinal ganglion cells—that is, the region of the columns of Goll. The more accurate our knowledge of the pathological anatomy of polyneuritis has become, the more frequently have we learned to recognize the involvement of the posterior columns of the cord in peripheral polyneuritic degeneration. In each individual case, therefore, it is hardly possible to decide with certainty to what extent certain symptoms (ataxia, disturbance of the muscular sense, slight bladder disturbances, etc.) are due to an involvement of the spinal cord or to that of the peripheral nerves. [Pseudo-tabes and true tabes sometimes coexist.—K.]

The *course* of this pseudo-tabes may extend over several years, but the prognosis is far more favorable than that of true tabes; decided improvement and even, in mild cases, complete recovery may occur; but there are also severe incurable forms, since dangerous complications (tuberculosis, etc.) often set in.

All the more acute forms of alcoholic neuritis afford quite a good *prognosis*, provided the abuse of alcohol can be permanently checked. We may then often see complete recovery in a few weeks or months. On the other hand, relapses are common, and these usually have a tedious course.

The first task in the *treatment* of alcoholic neuritis is to insist upon the absolute abandonment of the use of alcohol. If this be done, the milder cases, if properly taken care of, usually get well of themselves. The favorable course is aided by electrical (galvanic) treatment and tepid salt baths. Internally we prefer strychnin preparations (pills of nitrate of strychnin or extract of nux vomica). [In the severer cases with toe-drop, care must be taken to prevent contracture of the antagonist muscles (gastrocnemius and plantar muscles). If this contracture be allowed to develop it may greatly delay recovery. The timely use of splints and massage will prevent this.—K.]

[4. Beri-beri—Endemic Multiple Neuritis

The peculiar disease of endemic origin, long known in India and Japan as *"beri-beri"* or *"kak-ke,"* has been found by Scheube to be in its clinical and anatomical relations a well-characterized multiple neuritis. Cases are occasionally seen in seaport towns in sailors arriving from Asiatic ports, and Putnam has described cases occurring in fishermen returning from the Newfoundland banks. Independent outbreaks of the disease have also occurred among the insane in asylums in Alabama, Arkansas, Dublin, and elsewhere. Men are more frequently affected than women, and colored races more than the white. The cause of the disease is still obscure. Pekelharing and Winkler attributed it to a specific staphylococcus. Insufficient or improper diet probably increases the susceptibility to the disease, but it is doubtful if an exclusive diet of rice or fish, as was once believed, can actually cause beri-beri. It is probable that the use of "polished" or machine-milled rice may be a factor in causing the disease. It is thought that a fungoid toxin, "arsin," exists in the pericarp of the rice, which may enter the grain in the machine methods of preparation. Bondurant in Alabama found a close connection between the outbreak of the disease and the use of a polluted water supply. In other cases there seems to be some association with malarial poisoning. Overcrowding and foul air favor its development. The disease may begin suddenly or insidiously, and it is often ushered in by moderate fever and gastrointestinal symptoms—anorexia, vomiting, abdominal pain, and diarrhea. Two forms of the disease—a "wet" and a "dry"—are usually recognized. The symptoms in the "dry" form are similar to those of ordinary multiple neuritis—pain, paræsthesia, tenderness, loss of power, with degenerative muscular atrophy, especially in the extensors of the foot, slight disturbances of sensation, and diminution or loss of patellar reflex. The "wet" form, which is commoner, often begins with dyspnœa and a rapid pulse, and œdema may soon follow. The œdema is most marked in the regions supplied by the affected nerves, but there is also effusion into the serous cavities. The heart's action is weak, and the heart may soon become greatly dilated, and adventitious murmurs may be heard. In severer cases the urine contains a small amount of albumen, the dyspnœa and rapid pulse persist, and the patient succumbs to cardiac exhaustion. A certain amount of serous effusion is usually present at the autopsy, together with the changes in the heart. There is also a degenerative neuritis of the peripheral nerves, often including the vagus, and a corresponding degeneration of the muscles supplied by them, and often degeneration of the ganglion cells in the anterior cornua. In some cases of the "wet" form the disease progresses rapidly, with increasing œdema and cardiac weakness, leading to death in a week or two. Other cases may run a more chronic course, with atrophic paralysis, and eventually recover. The prognosis is grave. Twenty per cent of the cases among the chronic insane died in Alabama, and the mortality has been even greater in other epidemics, although Scheube estimates the average mortality at five per cent. In the way of prophylaxis good hygiene, a good soil and sanitary dwellings, pure water and plenty of nutritious food, seem essential. Cathartics, especially hydragogues, seem of some benefit in the early stages. The condition of the heart demands especial attention, but caution is necessary in administering digitalis or strophanthus (the chief drugs to employ) lest the weak heart be urged to undue effort. Hence we should begin with small doses (gm. 0.2 to 0.3, ♏iij to v, of the tincture), increasing gradually. In the later stages the treatment is the same as for other forms of multiple neuritis (strychnin, electricity, massage).—K.]

CHAPTER VI

NEW GROWTHS IN THE PERIPHERAL NERVES

THE new growths in the peripheral nerves are usually divided into false and true neuromata. True neuromata consist of newly formed, usually medullated, nerve fibers (*neuroma myelinicum* of Virchow), which are imbedded in a connective-tissue stroma, which is frequently very abundant. These neuromata develop most frequently in the cut ends of nerves in amputation stumps (amputation neuromata), but they may form after other injuries of the nerves, and perhaps many neuralgias and persistent pains after injuries are due to the formation of such little neuromata.

The so-called multiple neuromata or neuro-fibromata are of special pathological interest. They may be found in enormous numbers (many hundreds) on almost all the peripheral nerves, on the sympathetic plexuses in the abdominal cavity, and on the cranial nerves (vagus, etc.). The smallest of these tumors may be as large as a millet seed, the largest may grow to the size of a pigeon's egg or the thumb. The nerves are changed by them into thick cords covered with irregular enlargements which can be felt everywhere under the skin during life (on the nerves of the arm, the crural, the intercostal nerves, etc.). This remarkable condition can naturally be explained only by a congenital abnormal predisposition of the nervous system to tumor formation, a theory which is supported by a few observed cases of the hereditary occurrence of such multiple neuromata. The name "neuroma," however, is not correctly chosen. It is rather a fibroma (a tumor of connective tissue), through which the nerve fibers usually pass in good condition ("neurofibroma"). Hence it happens, as we have seen ourselves in one case, that, in spite of an extensive formation of neuro-fibromata, there are no nervous symptoms at all, no pain, no anæsthesia, no paralysis; but other cases have also been reported in which the tumors caused manifest and even severe symptoms.

Of other new growths on the nerves we may mention sarcoma. This also is often multiple, and then it may lead to very complicated types of disease. The so-called *tubercula dolorosa* deserve special mention. By this term we mean little nodules which may be felt beneath the skin, usually readily movable and very sensitive to pressure. They are not very rare, and are usually associated with drawing pains, which are rarely decidedly neuralgic and are not very strictly localized. They are situated in the extremities, especially in the arms, and in the back, the neck, etc. It is worthy of note that the symptoms are only at times very prominent, and then they disappear again, and that with this disappearance is certainly sometimes associated a spontaneous disappearance of the nodule. The anatomical nature of the tubercula dolorosa is not always to be established with certainty. Many of them are true neuromata, but others belong to other kinds of new growths.

The only successful treatment of neuromata is extirpation, which is to be undertaken only when the symptoms are very severe. If extirpation be not practicable, or if we have to do with multiple neuromata, the patient's trouble can be alleviated by symptomatic means, narcotics, and electricity. If we can compress the nerve above the neuroma, we can often cause by this means a temporary cessation of the pain.

II. THE DISEASES OF THE SPINAL CORD

CHAPTER I

DISEASES OF THE SPINAL MENINGES

1. ACUTE INFLAMMATIONS OF THE SPINAL MENINGES

Ætiology and Pathology.—Isolated acute inflammation of the spinal meninges is very rarely primary, so far as we know, but inflammatory processes in the neighborhood quite frequently involve the meninges, or a spinal meningitis occurs as one symptom of a general cerebro-spinal meningitis. This latter condition is seen chiefly in the idiopathic, generally epidemic, cerebro-spinal meningitis—a specific infectious disease, which has already been described in detail in a previous chapter. A tubercular spinal meningitis is also very often combined with tubercular inflammation of the cerebral meninges, but, since the symptoms of the latter are usually in the foreground of the picture, we will treat of tubercular cerebro-spinal meningitis in the section on diseases of the cerebral meninges. Secondary cerebro-spinal meningitis is sometimes seen in the course of certain other infectious diseases, and is then probably to be regarded as a special localization of the specific poison of the disease. This is the explanation of the occurrence of acute spinal and cerebral meningitis as a sequel of croupous pneumonia, and also of its occurrence in pyæmic and septic diseases, and, very rarely, in typhoid fever and the acute exanthemata. We must mention, finally, the occurrence of a purulent cerebro-spinal meningitis as a sequel of empyema, pulmonary gangrene, etc., which, although very rare, we have repeatedly noticed. In these cases the infection of the meninges also results from the primary foci of disease, but the channel of infection is not yet exactly known. Perhaps the intercostal nerves are the media of communication.

In all the cases so far mentioned we have chiefly an inflammation of the pia mater, a so-called leptomeningitis; the dura mater is not implicated in the disease at all, or only to a slight degree. The condition is different in those inflammatory processes which gradually invade the meninges from the neighboring parts outside the cord. Thus we very often see circumscribed inflammations on the outer surface of the dura (pachymeningitis) in caries of the vertebræ, and these inflammations often invade the inner surface of the dura, or more rarely reach the pia mater. Acute purulent peripachymeningitis is very rare; it is a purulent inflammation of the connective tissue between the dura mater and the vertebral column, which is always of secondary origin. We have seen a very characteristic case in the course of a puerperal pyæmia. The inflammation had spread from a purulent inflammation of the pelvic cellular tissue, through the foramina of the vertebral canal, and had finally set up a purulent inflammation on the outer surface of the dura, extending up to the cervical cord. We meet with an inflammation of the pia mater chiefly in diseases of the spinal cord, from an extension of the process, as the pia is involved to a greater or less extent in many cases of myelitis.

We do not know with certainty whether other influences, especially injuries and exposure to cold, can lead directly, as has often been claimed, to inflammation of the spinal meninges.

We need say but little in regard to the pathological anatomy of acute spinal meningitis. The changes in purulent inflammation of the pia mater have been described in the chapter on Epidemic Meningitis. Precisely the same conditions are found in the other forms of acute leptomeningitis. The changes in pachymeningitis are completely analogous. The dura mater is traversed by dilated vessels, and therefore is reddened; it is also thickened, and on its internal or external surface (*pachymeningitis interna* or *externa*, or *peripachymeningitis*) there is usually found a purulent or a seropurulent exudation.

Symptoms.—An accurate distinction between acute inflammations of the pia mater and those of the dura mater cannot be made clinically. The symptoms of the disease include the symptoms of any primary disease present, the general symptoms, such as fever, etc., and in addition the necessary consequences which the presence of a disturbance of the meningeal circulation and of the meningeal exudation exerts on the cord and nerve roots. These consequences are due both to a mechanical compression of the parts named, and often probably to an invasion of the substance of the cord itself by the inflammation. To these is added the frequent combination of spinal symptoms with the symptoms of a coexisting cerebral meningitis.

The symptoms of acute spinal meningitis are already known to us from the description of epidemic meningitis (see Vol. I, page 126). Recapitulating them briefly, we may mention chiefly the very severe pain in the back, the great sensitiveness of the vertebral column, and the stiffness of the back and neck. To these may be added usually symptoms of irritation on the part of the nerve roots—shooting pains in the trunk and the extremities, hyperæsthesia of the skin and of the deeper parts, symptoms of direct or reflex motor irritation, muscular tension, contractions, etc. The cutaneous and tendon reflexes are often, but not always, much diminished or entirely abolished in consequence of the lesion of the nerve roots. There are at times disturbances in the passage of urine and fæces. If, in the later course of the disease, there are actual paralysis and anæsthesia, they are usually a sign of a more marked implication of the cord itself.

Diagnosis.—From the symptoms named we can in many cases make a diagnosis of spinal meningitis. Of course a meningitis is found often enough on the autopsy table where the symptoms during life were completely obscured by other severe general symptoms, while, on the other hand, with severe constitutional symptoms, the symptoms of meningitis may be illusory, as in typhoid fever or pyæmia. Fuller information as to the seat and the extent of the inflammation is afforded by considering the most painful parts of the vertebral column, the predominance of pain and cutaneous hyperæsthesia in the arms (cervical region) or legs (lumbar region), etc. When the meningitis involves the upper portion of the cord and the medulla there may also be disturbances of respiration, symptoms in the pupils, and anomalies in the innervation of the heart. We can decide as to the form of the meningitis, whether purulent or tuberculous, only by a consideration of the history, the other morbid symptoms, and the course of the disease. Lumbar puncture is of great diagnostic assistance (*vide infra*, chapters on Tuberculous and Purulent Meningitis).

Prognosis.—We have seen an undoubted recovery, in severe cases, only in epidemic cerebro-spinal meningitis, and in the sporadic cases of idiopathic meningitis, which are probably identical in ætiology. In all other cases reported with a favorable termination the diagnosis may be doubted, for in general the rule is certain that, in extensive acute purulent leptomeningitis and pachymeningitis, the prognosis is almost absolutely unfavorable, whether it be secondary to another infectious disease or arise from propagation from some neighboring focus of inflammation. We may, perhaps, make an exception of certain mild, circumscribed cases, which do not come to suppuration, but these are also uncertain in regard to diagnosis.

Treatment.—In regard to treatment we must refer entirely to what has been said under epidemic and tubercular meningitis.

2. CHRONIC SPINAL LEPTOMENINGITIS

Although chronic leptomeningitis (usually wrongly termed chronic spinal meningitis) once played quite a large part in the diagnosis and pathology of diseases of the spinal cord, we must at present assert that its occurrence as an independent disease is exceedingly rare. Most of the reported cases of chronic meningitis were published at a time when the diagnosis of many diseases of the cord itself was still perfectly impossible, and when the thickenings and opacities of the meninges were much more striking at the autopsy than far more essential changes in the substance of the cord itself, which could be made out only by a careful microscopic examination, and not by the naked eye. In recent years only a very few cases have been published which can be regarded, at least with some reason, as primary chronic meningitis. It is especially hard to determine this, since syphilitic disease of the spinal meninges (*vide infra*) shows almost precisely the same anatomical appearance. Clinical experience is also entirely against the idea of the frequent occurrence of chronic spinal meningitis. Among many cases of spinal disease we shall scarcely be induced even to assume the probability of the existence of primary chronic meningitis.

The case is different with secondary chronic leptomeningitis. This, in the first place, is, in rare cases, the termination of an acute meningitis. Secondary leptomeningitis may certainly be detected, especially in epidemic meningitis. We also find chronic meningitis frequently as a secondary affection in primary diseases of the cord and the vertebræ. Thus, for example, in old cases of chronic spinal disease, associated with atrophy, such as tabes dorsalis, progressive muscular atrophy, etc., the pia is almost always quite opaque, thickened, and often united to the cord and the dura by very many firm adhesions, while a cloudy serogelatinous exudation is found in the meshes of the arachnoid. All these anomalies, however, are of a secondary nature, and have no clinical significance; for the same changes, though rarely so marked, are quite often found in old people, where they are analogous to the equally frequent opacities of the cerebral meninges, the pleuritic adhesions, etc., and where, during life, they have not caused the slightest symptoms of spinal disease.

The *symptoms* which have been set down as characteristic of leptomeningitis correspond to those of acute meningitis, except, of course, that they are relatively less intense, and that the course of the disease is more protracted. Pain and stiffness in the back and neck, abnormal painful sensations and paræsthesia in the extremities, a girdle sensation, and finally paresis, anæsthesia, and vesical disturbances, are the leading features of the type of disease as constructed, in whose fabrication there have been, at any rate, many confusions with myelitis, spinal syphilis, spondylitis, beginning tabes, multiple neuritis, etc. In the few cases confirmed by autopsy the clinical picture resembled in some degree, from the intention tremor of the arms and the spastic symptoms in the legs, that of multiple sclerosis. It is strange that often there were scarcely any shooting pains during life in spite of marked meningeal changes.

It is clear that under such circumstances no special rules for the *diagnosis* or even the *treatment* of chronic spinal meningitis can be given. Given a case, we would try local applications to the vertebral column; painting with iodin; dry, or, exceptionally, in strong patients, wet cups; also protracted tepid baths, 90° to 95° F. (26° to 28° R.), or cautious cold-water treatment; and, finally, the use of the galvanic current. Of internal remedies, iodid of potassium would be most indicated. We may refer to the description of the treatment of myelitis in regard to all further details.

3. PACHYMENINGITIS CERVICALIS HYPERTROPHICA

Pachymeningitis cervicalis hypertrophica was first fully described, as a special yet very rare form of disease, by Charcot in 1871, and later by his pupil Joffroy. Ætiologically, it is still utterly obscure. Alcoholism, exposure to cold, and injury have been alleged to cause it. No small number of cases seem to have some connection with syphilis.

Anatomically, the disease is characterized by a chronic and often very considerable thickening of the dura, almost always, as it seems, in the cervical portion of the cord, while the pia takes but a comparatively small part in the affection. The dura may attain a thickness of 6 or 7 mm., and usually appears composed of a number of concentric layers. Histologically, the hypertrophy consists of a new growth of dense connective tissue. The clinical symptoms of the disease arise from the fact that, first, the penetrating nerve roots, and later on the cord itself, undergo a considerable mechanical compression. If this is of high degree and persistent, there are, as a necessary result, secondary degenerations of the motor nerves and muscles, and a secondary descending degeneration of the pyramidal tract in the cord.

The clinical symptoms are easily understood from this. The disease almost always begins with severe pain, which shoots from the neck into the occiput and the arms. Besides this, there are paræsthesia and a numb feeling in the arms and hands. Rarely there is an eruption of herpes. All these symptoms depend upon the irritation of the posterior roots.

After this first period of the disease (*période douloureuse* of Charcot) has lasted some two or three months, the second period begins—the period of paralysis. An atrophic paralysis in the upper extremities gradually develops, mainly as a result of the compression of the anterior motor roots. This affects, in a remarkable manner, chiefly the distribution of the ulnar and median nerves—that is, the small muscles and flexors of the hand and fingers—while the distribution of the radial on both sides usually remains free. The hand, therefore, assumes a characteristic position (Fig. 145) as a result of the contracture of the antagonistic extensors. The paralyzed muscles rapidly become atrophic and show a marked reaction of degeneration. In this stage there may also be partial anæsthesia of the skin.

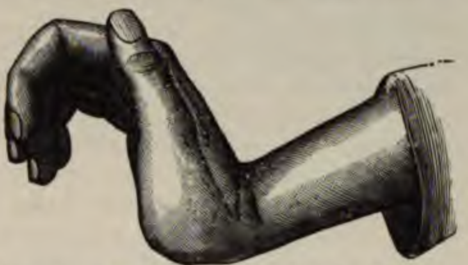


FIG. 145.—Position of the hand in pachymeningitis cervicalis hypertrophica. (From CHARCOT.)

If the compression of the cord advances, the motor fibers for the lower extremities, which pass through the cervical cord, must necessarily at last be involved sympathetically—the third period of the disease. The result of this is a spastic paralysis of the lower extremities—that is, a paresis or paralysis with increased tendon reflexes, but of course without muscular atrophy, because the trophic centers for the muscles of the legs, in the anterior cornua of the lumbar cord, remain perfectly intact. The compression of the cervical cord, however, may finally lead also to anæsthesia of the lower extremities, to paralysis of the bladder, and bedsores, under which symptoms death ensues; but, on the other hand, it must be mentioned that probably cases of recovery, or at least of actual improvement in pachymeningitis cervicalis hypertrophica, may occur even after it has lasted for years.

The diagnosis of the disease is based first upon the fact that the affection begins with pains in the arms, and upon the later appearance of the characteristic paralyses. It may easily be confused with tumors in the cervical cord and with cervical spondylitis. Amyotrophic lateral sclerosis is distinguished, however, by the absence of disturbances of sensibility, by the bulbar symptoms, and by the fact that the functions of the bladder remain intact.

Treatment can do little directly, and it must be chiefly symptomatic. Baths, sweating, iodid of potassium, and electricity are most used. Joffroy recommends the application of the actual cautery to the neck.

4. HEMORRHAGE INTO THE SPINAL MENINGES

(*Hematorrhachis. Meningeal Apoplexy. Pachymeningitis spinalis hemorrhagica interna*)

Large hemorrhages into and between the spinal meninges are of rare occurrence. They arise chiefly from traumatic influences, from concussion or fracture of the vertebral column, or from direct injuries of the meninges, such as stabs or gunshot wounds. In a few cases great physical exertion may also lead to a meningeal apoplexy. Diseases of the vertebræ, caries, and carcinoma, may also lead to hemorrhage from the erosion of a vessel. The frequent little meningeal hemorrhages which appear as a complication of meningitis, in hemorrhagic diseases, in the course of severe general infectious diseases, septic infection, typhoid fever, and smallpox, and as a result of severe general convulsions, very rarely have any clinical significance. Finally, it may be mentioned that aneurism of the aorta or its branches may rupture into the vertebral canal.

The clinical symptoms of meningeal hemorrhage are almost always sudden and "apoplectiform," but are unattended by any disturbance of consciousness. Their intensity depends entirely upon the degree of compression which the nerve roots and the cord undergo from the effused blood. The symptoms of irritation usually predominate—severe pain in the back, paræsthesia, and neuralgic pains in the extremities; and also symptoms in the motor distribution, tension, tremors, and contractures of the muscles. With large hemorrhages, symptoms of paralysis, partial anæsthesia, disturbances of the bladder, symptoms of unilateral lesion, etc., may ensue, but the different types of the disease of course vary with the seat of the hemorrhage. On the whole, the diagnosis of meningeal hemorrhage can but rarely be made with any certainty, unless suggestive ætiological factors precede and the symptoms and manner of beginning are specially characteristic.

The course is quite favorable in many cases if the blood be rapidly reabsorbed, but sometimes a permanent disturbance of function is left.

In regard to treatment, complete rest and the local use of ice are chiefly to be recommended, and also local bloodletting—dry cups, or leeches, where there are severe initial symptoms of irritation. Under some circumstances we may try to remove a part of the blood by lumbar puncture, and thus relieve the pressure on the cord. [When the diagnosis of hemorrhage into the meninges is reasonably certain, an attempt should be made to relieve the condition by opening the spinal canal and removing the clot.—K.] If permanent disturbances be left, they should be treated by the ordinary methods—iodid of potassium, baths, and electricity.

We must here speak of *pachymeningitis interna hemorrhagica* as a special form of disease, which usually occurs at the same time with hematoma of the cerebral dura mater (*vide infra*), and is precisely analogous to it in its ætiology and pathological anatomy. Encapsulated collections of blood are found on the inner surface of the dura; these often have a considerable circumference, and contain blood already disintegrated, detritus, hematoidin crystals, etc., since they are usually of old standing. Besides this, there are also the signs of a fibrinous inflammation—as in the cerebral dura—which, according to the opinion of most observers, is the primary process, so that the hemorrhages into the newly formed false membrane are secondary. The symptoms of the affection—which has been observed chiefly in the chronic insane (general paralytics) and drunkards—are rarely pronounced, and consist chiefly of pain in the back, stiffness of the vertebræ, and some signs of compression on the part of the nerve roots and the cord, but we can very rarely make a definite diagnosis.

CHAPTER II

PRELIMINARY REMARKS ON THE LOCALIZATION AND TOPICAL DIAGNOSIS (SEGMENTAL DIAGNOSIS) OF DISEASES OF THE SPINAL CORD

THE pathological processes that take place in the spinal cord may be divided into two groups, according to their localization. In the first group the anatomical changes are limited with remarkable regularity to definite neuron territories, which are associated both anatomically and physiologically. These diseases are called *systemic diseases,* since the entire group of functionally associated neurons is called a "system." We may, in all probability, conceive that these affections arise in one of two ways—either certain neuron systems in some individuals have from the outset a congenital (inherited) weak and morbid predisposition, and, in consequence thereof, they are not able to perform their functions, but atrophy prematurely; or that certain external, usually toxic, influences exert their morbid action, not on the whole nervous system or fortuitous circumscribed parts thereof, but only on certain definite neurons (cells or fibers)—"elective injuries." The latter hypothesis finds a fitting and instructive example in the behavior of many well-known poisons (curare, strychnin, morphin, lead, etc.), which injure only certain very definite neurons and parts of nerves, while they leave all other neurons completely intact. According as the disease involves one or more neuron systems we speak of *simple* or *combined* system diseases. Spinal muscular atrophy, amyotrophic lateral sclerosis, etc., are due to disease of the motor neurons in the corticomuscular tract; they are simple system diseases. Hereditary ataxia, on the other hand, is an example of a combined system disease, since in this usually not only certain peripheral sensory neurons but also motor neurons undergo simultaneous degeneration. Since the processes of many neurons extend, on the one hand, from the spinal cord or spinal ganglia into the peripheral nerves, and, on the other, from the cord to the brain, and *vice versa,* we cannot, strictly speaking, class the systemic neuron diseases with diseases of the spinal cord in their limited sense. Only for practical and, in part, purely conventional reasons do we consider the systemic diseases in this section.

In distinction from the *systemic diseases* there is a second group of diseases of the spinal cord where either there is no such limitation of the anatomical process to definite neuron territories, or the limitation is only very slight. In these cases the disease extends a greater or less distance transversely and longitudinally in the cord, forming either one large focus of disease of irregular outline or many smaller, irregularly disseminated foci. To this group, the *unsystemic, diffuse diseases* of the spinal cord, belong all the traumatic destructions, also the hemorrhages, new growths, and inflammations of the spinal cord (myelitides), multiple sclerosis, etc.

The diagnosis of diseases of the spinal cord is usually, first of all, a topical diagnosis. From the symptoms present in the individual case we try to determine first that place in the spinal cord where an affection must be situated to explain the given symptoms. By appraising all the existing symptoms of disease, and also all the functions that are still in normal activity, we can determine whether the disease is limited to one definite neuron system or whether it must spread diffusely over a larger portion of the spinal cord. Only after the topical diagnosis of the disease is thus established do we try to form a judgment as to the nature of the disease by connecting it with well-known typical forms of disease or by considering the whole course of the disease and any accompanying symptoms.

The topical diagnosis naturally presupposes an accurate knowledge of the functions of all the different parts of the spinal cord—a knowledge which at present we by no means possess, but we know at least the distribution of the motor and sensory functions in the different portions of the cord in their main outlines, and upon this basis we can often determine, at least with approximate certainty, the localization of the disease, the so-called segmental diagnosis, in the transverse lesions of the spinal cord.

1. **Motor Functions.**—In regard to the localization of motor functions we know definitely that the motor fibers of the anterior roots all arise from the ganglion cells of the anterior cornua. To each individual muscle belongs a group of cells in the anterior cornua of the cord, from which the corresponding motor nerves, which innervate the muscle, arise. These different muscle nuclei, however, apparently do not lie simply beside and above one another, but they encroach upon one another, so that naturally their accurate definition becomes difficult. Furthermore, the fibers of one muscle nucleus do not always pass out from a single anterior root, but often from two or more roots. The following summary of the arrangement of the spinal muscle nuclei in the different segments of the spinal cord has been compiled by the aid of the well-known tables of Allen Starr, Kocher, Bruns, and others, but it can be regarded as only an approximate expression of the actual conditions. Many details will be changed and many gaps filled by later investigations. The muscles printed in italics derive the main part of their nerves from the corresponding spinal segment:

Segments or Anterior Roots	Muscles
1st Cervical Segment.	*Small muscles of the neck.* (Obliquus capitis superior et inferior, rectus capitis posticus major et minor.) *Sternohyoideus.* *Sternothyroideus.* } Ramus descendens hypoglossi. *Omohyoideus.* Thyreohyoideus. Geniohyoideus.
2d Cervical Segment.	Rectus capitis posticus major. Longus colli. Complexus major et minor (trachelomastoideus). Cervicalis ascendens. Splenius capitis et colli. *Sterno-cleido-mastoideus.* *Trapezius.*
3d Cervical Segment.	Longus colli. Complexus. Cervicalis ascendens. Splenius. Trapezius.

SEGMENTS OR ANTERIOR ROOTS (*Continued*)	MUSCLES (*Continued*)
4th Cervical Segment.	Splenius, trachelomastoideus, longus colli. *Scaleni.* *Diaphragma* (N. phrenicus). Levator scapulæ. Supraspinatus. Infraspinatus. Deltoideus. Biceps, supinator longus. Rhomboidei.
5th Cervical Segment.	*Rhomboidei* (N. dorsalis scapulæ). *Supra- et infra-spinatus* (N. suprascapularis). *Deltoideus* (N. axillaris). *Biceps.* *Coracobrachialis.* } N. musculo-cutaneous. *Brachialis internus.* *Supinator longus et brevis.* Diaphragma. Pars clavicularis pectoralis majoris. Serratus anticus major. Teres minor. Latissimus dorsi.
6th Cervical Segment.	*Pectoralis major et minor.* *Latissimus dorsi.* *Subscapularis.* *Teres major.* *Serratus anticus major* (N. thoracicus longus). Triceps. *Pronator teres et pronator quadratus.* Biceps, brachialis internus. Pars clavicularis pectoralis majoris. Extensors and flexors of the hand. Scaleni.
7th Cervical Segment.	*Triceps.* *Extensors of the hand.* (Radiales externi, s. extensor carpi radialis longus et brevis, ulnaris externus, s. extensor carpi ulnaris.) *Extensors of the fingers.* (Extensor communis digitorum, extensor pollicis longus et brevis, extensor indicis.) Flexors of the hand. Pronators of the forearm. Portio sternocostalis pectoralis majoris. Latissimus dorsi. Subscapularis, teres major.
8th Cervical Segment.	*Flexors of the hand.* *Long flexors of the fingers.* (Flexor digitorum sublimis et profundus, flexor pollicis longus.) Small muscles of the hand,

SEGMENTS OR ANTERIOR ROOTS (*Continued*)	MUSCLES (*Continued*)
1st Dorsal Segment.	*Small muscles of the hand and fingers.* (Interossei, thenar, hypothenar.) Long extensors of the thumb. *Dilatator pupillæ* (to N. sympatheticus).
2d to 12th Dorsal Segment.	Back muscles.
2d to 11th Dorsal Segment.	Intercostal muscles.
7th to 12th Dorsal Segment.	Abdominal muscles.
1st Lumbar Segment.	Lowest abdominal muscles. *Quadratus lumborum.* Iliopsoas. *Sartorius.*
2d Lumbar Segment.	*Cremaster.* *Iliopsoas* (psoas, iliacus internus).
3d Lumbar Segment.	Iliopsoas. *Adductors* of the thigh. *Pectineus.*
4th Lumbar Segment.	*Extensor cruris quadriceps.* Obturator externus (?) Gracilis, adductores. Tibialis anticus.
5th Lumbar Segment.	*Glutæus medius et minimus.* *Semimembranosus, semitendinosus.* *Biceps femoris.* Tensor fasciæ latæ. Dorsal extensors of the foot and toes.
1st Sacral Segment.	*Pyriformis.* *Obturator internus.* *Gemelli.* *Quadratus femoris.* } Outward rotators of the thigh. *Dorsal extensors of the foot and toes.* (Tibialis anticus, peronei, extensor digitorum communis.)
2d Sacral Segment.	*Flexors of the foot* (gastrocnemius, soleus). Flexor digitorum communis, flexor hallucis longus. Tibialis posticus. Small muscles of the foot. *Erection.*
3d Sacral Segment.	*Perineal muscles.*
4th Sacral Segment and Coccygeal Segment.	Striated muscles of the urethra, the rectum and the genital organs.

Considering the muscles with regard to their function, we obtain the following table of the motor functions of the spinal segments:

PLATE VI

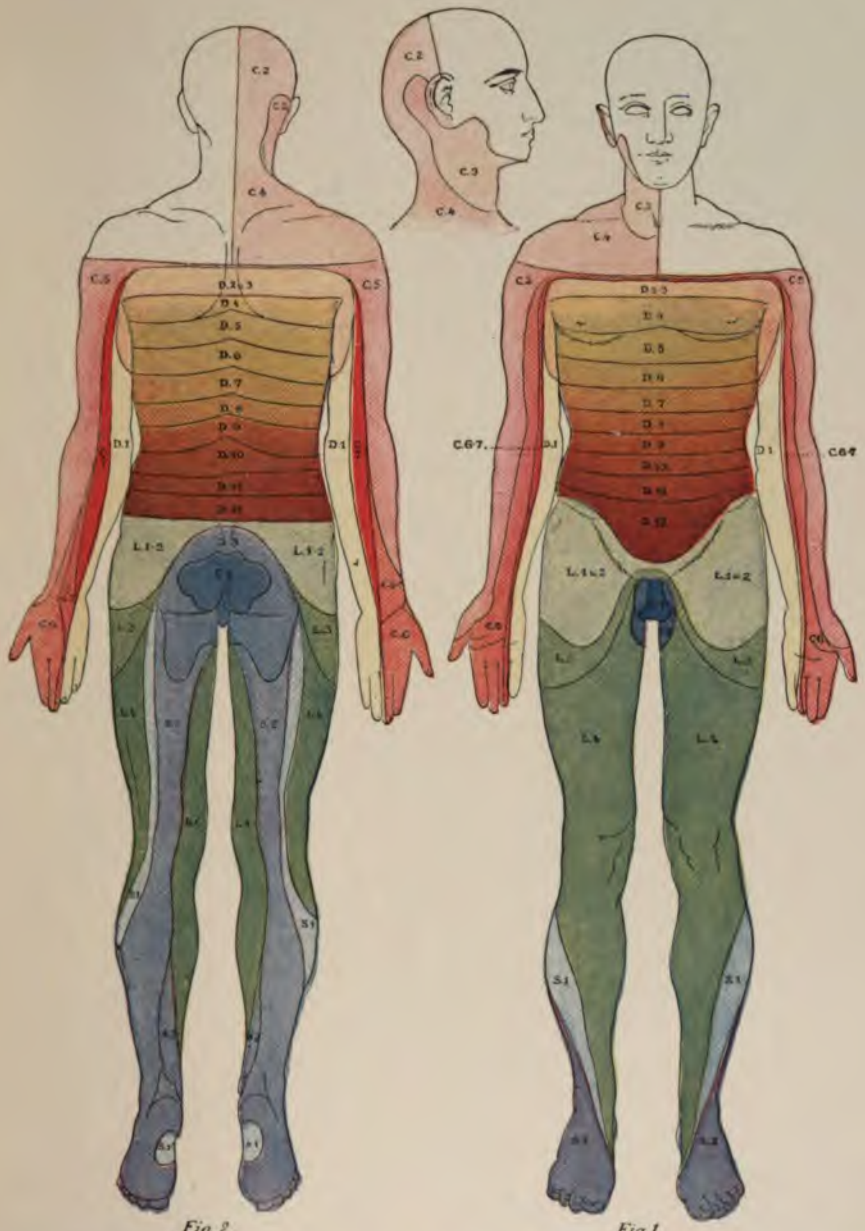


Fig. 2. *Fig. 1.*

Distribution of the Areas of the Sensory Roots upon the Surface of the Body (from Kocher).

Red: Area of the cervical roots (C. 2 to C. 7).
Yellow: Area of the dorsal roots (D. 1 to D. 12).
Green: Area of the lumbar roots (L. 1 to L. 4).
Blue: Area of the sacral roots (S. 1 to S. 4).

Cervical Cord

1st Cervical Segment: Turning the head. Bending the head backward.
2d and 3d Cervical Segments: Bending the head forward. Raising the shoulders.
4th Cervical Segment: Inspiration. Drawing the shoulders backward.
5th Cervical Segment: Raising and outward rotation of the upper arm. Flexion and supination of the forearm.
6th Cervical Segment: Adduction and inward rotation of the upper arm. Extension and pronation of the forearm.
7th Cervical Segment: Flexion and extension of the hand.
8th Cervical Segment: / *1st Dorsal Segment:* } Flexion and extension of the fingers. Movement of the thumb.

Lumbar Cord

2d and 3d Lumbar Segments: Flexion and adduction of the thigh.
4th Lumbar Segment: Extension of the lower leg.
5th Lumbar Segment: Abduction and inward rotation of the thigh. Flexion of the lower leg.
1st Sacral Segment: Extension and outward rotation of the thigh. Dorsal extension of the foot.
2d Sacral Segment: Plantar flexion of the foot. Toe muscles.

If there is disease of the anterior horns of the gray matter (poliomyelitis, syringomyelia, hematomyelia) or compression of the anterior motor roots, the muscles paralyzed will correspond directly to the affected segments; but with more extensive transverse disease (myelitis, tumors, compression) of course those msucles will also be paralyzed whose nuclei lie below the seat of the lesion, since the fibers of the pyramidal tract leading to them will be interrupted. From what has already been said (page 199), we may readily distinguish the nuclear or root paralyses from the pyramidal (lateral) tract paralyses by the greater atrophy, and especially by the electrical reaction of degeneration.

2. **Sensory Functions.**—The distribution of the sensory conducting path to the different segments of the spinal cord corresponds in general to the territories of distribution of the sensory fibers arising from each spinal ganglion (*vide* page 152 *et seq.*); but since the outgoing sensory fibers are mixed with one another in the plexuses, each region of the skin is supplied by sensory fibers from at least two, and probably three, spinal segments, or from two or three posterior spinal roots. The accompanying diagram, devised by Kocher (Plate VI), gives, as far as our present knowledge admits, a good representation of the extent of the different root territories. We must be prepared for individual variations. We see that in the arms the radial region is innervated from the upper (fourth and fifth) roots of the brachial plexus and the ulnar region from the lowest roots (first, and probably second, dorsal roots). If we conceive of the arm raised horizontally with the thumb directed upward, we can in a way imagine a horizontal segmental division of the arm according to the different sensory-root areas. On the trunk the segmental arrangement of the different sensory-root areas is very plain, although the segments do not run parallel to the ribs and intercostal nerves, but they are strictly horizontal. However, the upper borders of spinal anæsthesias generally extend a little farther up on the back than on the anterior surface of the body.

In the legs Kocher's diagram fails to give the area of distribution of the fifth sensory lumbar root. We therefore add for comparison the diagram

given by Thorburn for the distribution of the sensory-root areas from the lumbosacral plexus (Fig. 146). The two diagrams differ considerably from each other, so that further investigations are very desirable. Figs. 147 to 153 (from work conducted in the author's clinic by L. R. Müller) show summarily the distribution of the anæsthetic (shaded) areas of the skin in transverse lesions of the spinal cord at different levels of the lumbosacral cord.

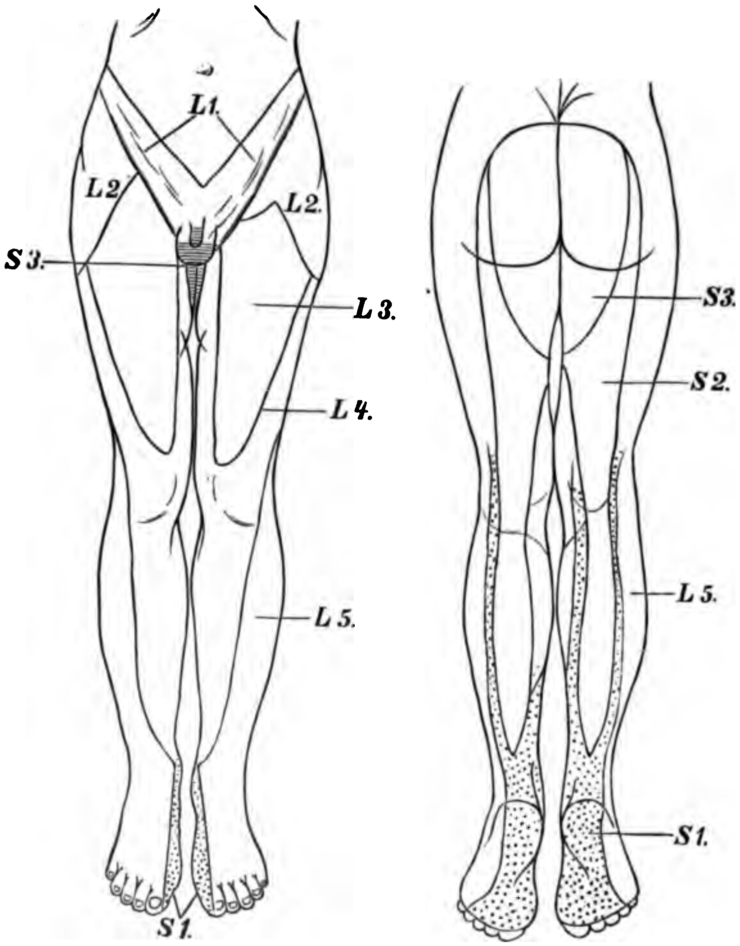


FIG. 146.—Distribution of the sensory-root areas from the lumbosacral plexus (from Thorburn).

In subsequent chapters we will return to many details in regard to the significance of disturbances of sensation in accurate segmental diagnosis. We can here speak of only two important points. Symptoms of sensory irritation are regarded in spinal diseases almost exclusively as root symptoms, due to an irritation of the affected posterior roots by pressure, laceration, etc. Such symptoms of irritation, therefore, are seen chiefly in diseases which arise in the coverings of the spinal cord (the vertebræ and the spinal meninges).

Then we must also refer once more to the circumstance mentioned on page 156, that the tracts for the conduction of sensations of pain and temperature after their entrance into the cord probably go at once to the gray matter of the posterior horns, while the simple sensations of touch, pressure, and position are conducted to the brain through the white posterior columns. If we find, therefore, in spinal diseases the so-called dissociated anæsthesia, especially anæsthesia for pain and temperature with retained sensibility for touch and position, we may be quite certain that there is a central lesion in the posterior gray cornua, such as is found especially in syringomyelia, injury of the cord, and central glioma.

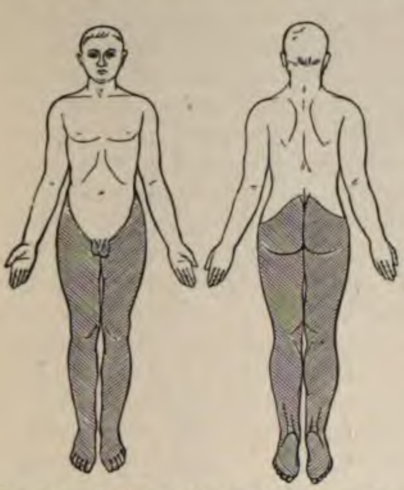


FIG. 147.—Lesion at the level of the second lumbar segment.

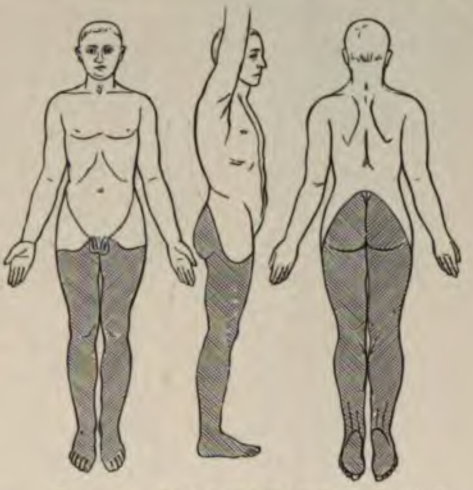


FIG 148.—Lesion at the level of the third lumbar segment.

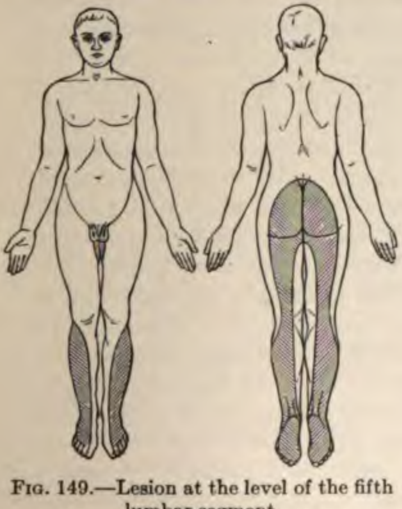


FIG. 149.—Lesion at the level of the fifth lumbar segment.

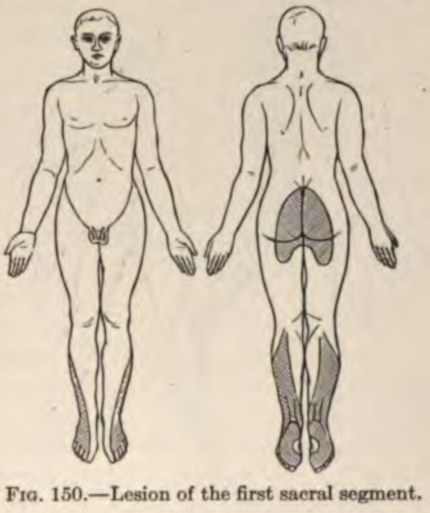


FIG. 150.—Lesion of the first sacral segment.

[Brissaud and other recent French writers lay much stress upon the difference between the sensory areas supplied by the spinal roots (radicular metamerism) and the sensory areas supplied by the different spinal segments (spinal metamerism). The former have already been depicted. It is claimed that the latter areas surround the limb, their boundaries being perpendicular to its long axis. They correspond, therefore, to areas covered by a glove, a sleeve, a stocking, etc. (geometrical anæsthesia). The determination of the areas of anæsthesia may therefore be of importance in deciding whether disease affects merely the spinal roots or the substance of the cord. Dejerine, however, denies any spinal metamerism, and various German observers claim that in syringomyelia, where the central gray matter is affected, the areas of anæsthesia correspond to the root distribution as given above, and are not arranged in bands about the limb; that there is no difference between radicular and spinal metamerism, and that lesions of any spinal segment cause practically the same distribution of anæsthesia whether the roots or the central gray matter be involved. The question is still unsettled, and further study is necessary.—K.]

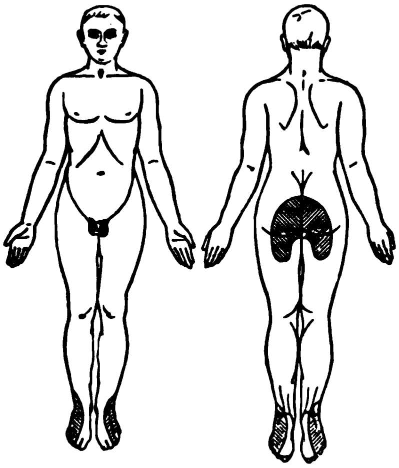


Fig. 151.—Lesion of the second sacral segment.

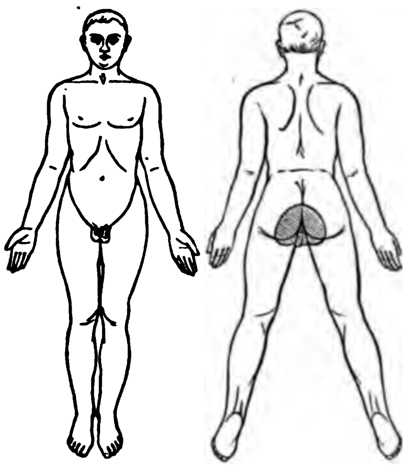


Fig. 152.—Lesion of the third sacral segment.

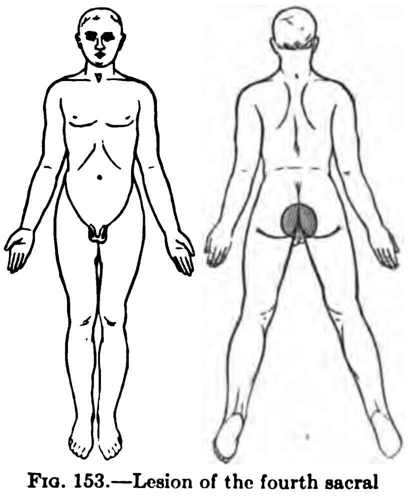


Fig. 153.—Lesion of the fourth sacral segment.

3. **Reflexes.**—The presence or absence of the reflexes may also aid the segmental diagnosis. Of course the conditions here are often very ambiguous, since the reflex may be absent either from an interruption of its particular

centripetal or centrifugal roots or from an interruption in any part of the reflex arc itself. The centripetal and centrifugal portions of the reflex arc are not always necessarily at the same level. Our knowledge is by no means absolutely definite as to the position of the reflex arc itself. The statements as to the relation of definite segments of the cord to certain reflexes therefore refer in general only to afferent (centripetal) posterior nerve roots involved in such reflexes. The assumption that the transmission of the irritation to the appropriate motor ganglion cells takes place in the same corresponding segment of the spinal cord, is warranted at most for certain simple reflexes (patellar reflex, etc.). For more extensive reflexes (general flexion reflex in the leg, etc.) we must assume from the outset that the reflex process spreads over many segments of the spinal cord.

We have set forth the relations of the spinal segments (or posterior roots) to the different reflexes in the following table, which represents our present knowledge of the subject, although it is still very uncertain:

Biceps tendon reflex.	5th cervical segment (?)
Triceps tendon reflex.	6th cervical segment (?)
Tendon reflexes in the forearm.	7th and 8th cervical segments (?)
Upper abdominal reflex.	8th and 9th dorsal segments.
Middle and lower abdominal reflex.	10th to 12th dorsal segments.
Cremaster reflex.	1st to 3d lumbar segments.
Patellar reflex.	2d to 4th lumbar segments.
Glutæal reflex.	4th and 5th lumbar segments.
Plantar flexor reflex.	1st and 2d sacral segments.
Achilles tendon reflex.	1st and 2d sacral segments.
Anal reflex.	5th sacral segment.

Many details as to the behavior of the reflexes in different spinal diseases will be mentioned in later chapters. We may here state briefly that in general the absence of a reflex points to a lesion of the corresponding segment of the spinal cord. Transverse diseases above any given reflex arc are usually (but not always, *vide infra*) associated with a pronounced increase of the reflex. In diseases of the lumbar cord the tendon reflexes in the lower extremities are therefore lost, as a rule, while in the diseases of the dorsal and cervical cord they are usually much increased.

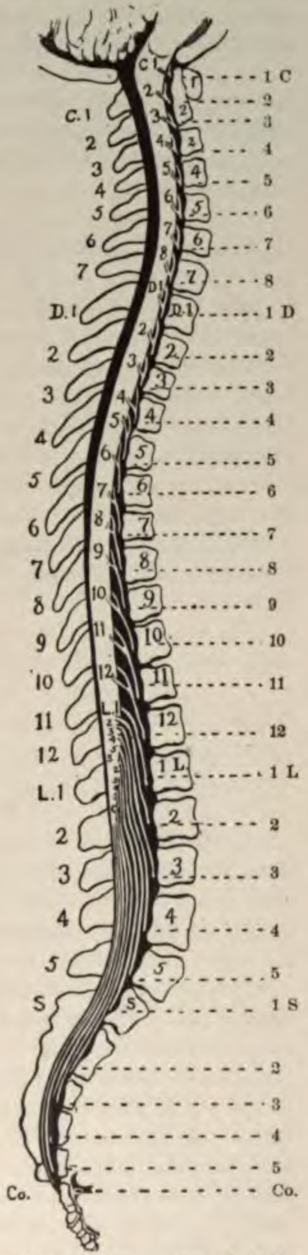


Fig. 154.—Diagram to explain the relation between the points where the different spinal roots are given off from the cord and the different vertebral bodies and spinous processes.

4. **Anatomical Relations.**—At this point we must mention some important anatomical relations between the position of the different portions of the spinal cord and the surrounding vertebræ. It is well known that the lower end of the spinal cord extends only to the level of the second lumbar vertebra. The vertebral canal below this point is filled partly by the filum terminale, but chiefly by the bundles of roots of the cauda equina. The greatest breadth of the cervical enlargement is at the level of the fifth and sixth cervical vertebræ. The dorsal cord begins at the level of the second dorsal vertebra, the lumbar cord at the level of the tenth dorsal vertebra. The position of the conus terminalis corresponds to the upper half of the second lumbar vertebra. The topical relations between the different spinal segments and spinal roots and the bodies of the vertebræ are also of great practical importance. Since the spinal segments are shorter in the vertical direction than the bodies of the vertebræ, the spinal roots, from above downward, must travel farther and farther downward to reach their corresponding intervertebral foramina. The accompanying diagram from Gowers (Fig. 154) makes this relation plain. A lesion of the eighth dorsal segment, for example, lies opposite the body of the seventh, and not of the eighth, dorsal vertebra; but since the spinous process also has a descending direction, the lesion must be sought at the level where we can feel the seventh or even the sixth spinous process. These relations have great practical significance for any operative procedure, but we must also remember that there may be individual variations from the rule.

CHAPTER III

DISTURBANCES OF CIRCULATION, HEMORRHAGES, TRAUMATIC LESIONS, AND FUNCTIONAL DISTURBANCES OF THE SPINAL CORD

1. **Disturbances of Circulation.**—Our knowledge as to the occurrence and as to the clinical significance of pure disturbances of circulation in the spinal cord is very slight. Most that is stated in regard to them in the older descriptions of the pathology of the spinal cord corresponds much more to theoretical hypotheses than to the actual objective facts.

It goes without saying that a complete anæmia of the spinal cord must destroy its function; this fact is best illustrated by the well-known experiment of Stenson: if we compress the abdominal aorta of an animal, and thus cut off the blood supply to the lumbar cord almost completely, a paralysis of the posterior portion of the body very rapidly ensues. Embolic (or thrombotic) obstruction of the aorta also leads to flaccid paralysis of the legs in man, but this paraplegia is to be referred to anæmia of the peripheral nerves and muscles, and not to any change in the spinal cord, as we must maintain after a personal observation upon this point. The spinal cord is abundantly supplied with blood by the spinal arteries coming from the vertebral artery. Some precisely analogous observations have been made in man in the rare cases of obstruction of the aorta by emboli or thrombi. Pronounced spinal symptoms in general anæmia, which may be referred to a coexisting anæmia of the cord, are rare, and at any rate are of much less clinical prominence than the important results of a coexisting cerebral anæmia (*vide infra*). In only a few cases has paraplegia been seen after a great general loss of blood, as after metrorrhagia or intestinal hemorrhage. The severe spinal symptoms sometimes occurring in pernicious anæmia are due to combined columnar degeneration in the cord (*vide infra*).

All statements as to the occurrence and clinical significance of hyperæmia of the spinal cord are still more dubious. We do not know whether there is active hyperæmia of the cord. The hyperæmia from stasis, in general disturbances of the circulation, in which certainly the spinal cord often takes part, causes no especially marked symptoms.

2. **Hemorrhage Into the Substance of the Spinal Cord—Spinal Apoplexy—Hematomyelia.**—Primary hemorrhage into the spinal cord is as rare as hemorrhage into the brain is common. It is due most frequently to traumatic influences (fall on the buttocks, blow on the back, etc.). We will return to this traumatic hematomyelia later. The sudden onset of spinal paralysis has also been seen after great physical exertion, and this is probably due to a spinal apoplexy. It is possible, but it is not yet absolutely certain, that primary vascular disease (small aneurisms) may favor the occurrence of hemorrhage in such cases. Small capillary spinal hemorrhages are not infrequently seen as a secondary complication in tumors of the cord, and in inflammatory affections such as myelitis, epidemic meningitis, etc., and in the general hemorrhagic diathesis, as in scurvy or severe general infectious diseases, and after great congestion, convulsions, etc. F. Schultze has also found hemorrhages into the gray matter of the posterior horns in newborn infants who have come into the world asphyxiated after difficult labor, and these hemorrhages may perhaps be of some significance in later life.

Anatomical experience in regard to primary spinal apoplexy is still quite slight. The commonest situation for spinal hemorrhage is the gray matter, either in the cervical or the lumbar enlargement. If the hemorrhage be abundant, we find the substance of the cord destroyed to a great extent. The apoplectic center usually extends principally in the long axis of the cord. The blood is still fluid in fresh cases. Later on it undergoes all those changes which are fully described in the chapter on Cerebral Apoplexy. It is not improbable that some cases of syringomyelia may be referred to a primary hemorrhage into the spinal cord.

The *symptoms* of spinal apoplexy must in the first place depend entirely upon the seat and extent of the hemorrhage. The sudden, apoplectiform onset of the symptoms is always characteristic. It usually begins with severe pain in the back, followed very speedily by more or less widespread paralysis, anæsthesia, vesical disturbances, etc. Since the hemorrhage is usually situated in the gray matter, the localization of the muscular paralyses and the form of sensory disturbance (dissociated anæsthesia!) often show the peculiarities attendant upon such a lesion. Hemorrhage into one half of the cord sometimes shows very definite symptoms of unilateral lesion (*vide infra*). Thus, in some cases of hemorrhage into the cervical cord, in particular, Minor has observed the following symptom-complex: partial atrophic paralysis of one arm, spastic paralysis of the leg on the same side, partial anæsthesia (just as in syringomyelia) in the extremities of the opposite side.

The *course* of hemorrhage of the cord may in many cases be comparatively favorable. If the blood be absorbed, and essential paths of conduction be not permanently destroyed, the symptoms of paralysis gradually pass away, and recovery or at least improvement and an arrest of the symptoms follow. In many cases, of course, the severe type of spinal paralysis develops, with bedsores, cystitis, etc., and this, after a longer or shorter time, leads to death.

We must always be very guarded in making a *diagnosis* of spinal hemorrhage. We can do so with some probability only when the symptoms begin in a pronounced apoplectiform manner, and when definite ætiological factors (trauma) are to be made out; but, as we shall see later, central softening without actual hemorrhage may follow trauma and cause precisely the same symptoms as hematomyelia. Very frequently injuries (fractures) of the bodies of the vertebræ are found to be present at the same time. We should also not forget that many forms of acute myelitis, and even chronic spinal affections (especially spinal syphilis), may also show a remarkably sudden onset, or at least may suddenly become worse. The distinction between genuine spinal apoplexy and meningeal hemorrhage is often difficult. Initial symptoms of marked sensory irritation favor meningeal hemorrhage, while the occurrence of symptoms pointing to an affection of the gray matter, especially the combination of atrophic muscular paralysis with partial anæsthesia (thermo-anæsthesia, analgesia), warrants the suspicion of central hematomyelia. The results of lumbar puncture may be of the greatest assistance in the diagnosis (presence of blood in the cerebro-spinal fluid, etc.).

Treatment.—If we have the rare opportunity to be able to interfere at the beginning of the symptoms, we should prescribe a perfectly quiet position in bed, local use of ice, and eventually ergotin. Later on the treatment should be directed according to the methods generally in use in spinal paralysis.

3. **Traumatic Lesions of the Spinal Cord.**—In spite of the protected position of the spinal cord, it is often the seat of severe acute traumatic lesions. Fractures and dislocations of the vertebræ are the most frequent, and these may give rise to considerable injury of the spinal cord by the dislocation of the vertebræ, or by the projection of a fragment of bone. The recent Röntgen-ray examinations have given us an idea of the frequency of fractures of the spine, especially of the milder varieties, such as separation of the transverse processes, etc. Many cases which, owing to the lack of all objective symptoms, were formerly classed as pure hemorrhage or traumatic softening, are shown by the Röntgen-ray examination to be injuries of the spinal column and their sequelæ. Dislocations occur most frequently in the cervical region (between the atlas and axis or very often between the fifth and the sixth cervical vertebræ). The upper vertebra in such cases is usually dislocated forward. The spinous process of the lower vertebra therefore projects backward, while the spinous process of the dislocated vertebra is pushed forward. The head is bent forward. Every movement of the head is painful and is anxiously avoided. Fractures are most frequently situated in the middle cervical vertebræ, the upper dorsal, and particularly at the twelfth dorsal and first lumbar vertebræ.

Besides the indirect lesions of the cord by displaced vertebræ, the action of the injury (fall on the back, etc.) may also extend immediately to the spinal cord. In many cases there may be at the moment of the injury a temporary distortion of the vertebral column associated with local laceration or contusion of the cord; but sometimes severe traumatic lesions of the cord occur without any vertebral injury. These consist of hemorrhages, which are usually central (hematomyelia, *vide supra*), or traumatic softening, which is also usually central. Hemorrhage and softening may also be associated. Of course the different sequelæ of the traumatic violence frequently occur in combination. Fractures are associated with dislocations of the vertebræ, and with these there may be found in addition traumatic meningeal or central hemorrhages, traumatic softening, etc. Thus, we not infrequently see, in particular, above and below the destruction of the cord caused by a vertebral lesion, central hemorrhages, or softening in the anterior and posterior horns.

Gunshot wounds of the cord are quite frequent, in which the bullet either penetrates the cord itself or produces injury of the vertebræ and hemorrhage, which involve the cord indirectly. Stabs and incised wounds of the cord have been frequently seen. The point of a knife or sword may penetrate the spinal canal from behind through the soft parts and cause a partial section or at least a contusion of the cord. In some of these cases, also, the conditions are often rendered very complicated by the coincident hemorrhage, the possible secondary traumatic inflammation, etc.

The *symptomatology* of injuries of the spinal cord follows the general rules for the localization of disease of the cord laid down in the previous chapter. There is usually at first a pronounced and often complete motor paralysis of the lower extremities. There is also anæsthesia, and vesical and rectal paralysis. In many severe cases the secretion of urine seems at first much diminished or wholly wanting. If the spinal roots be affected there are severe shooting pains and paræsthesia. Above the limit of total cutaneous anæsthesia we sometimes find a zone with dissociated anæsthesia (analgesia and thermo-anæsthesia). This symptom points to a hematomyelia or a central softening in the corresponding posterior cornu of the gray matter above the seat of the special lesion (*vide supra*). The reflexes are usually diminished at first, but, later, if the injury be above the reflex arc, they are increased; but if the arc itself be broken, they are permanently absent. This rule, however, undergoes an interesting exception, since we often find after severe injury of the cervical cord, and especially after a complete transverse lesion thereof, a flaccid paralysis of the lower extremities and complete absence of the tendon, and more rarely of the cutaneous, reflexes (Bastian, Thorburn, Bruns, and others). To explain this striking fact we must suppose that the reflex path in the lumbar cord has also undergone some disturbance, either from the initial general concussion or from its separation from all the higher parts in the brain. With the loss of reflex is often associated paralysis of the bladder and rectum. We often see in men, especially after injury of the cervical cord, a more or less complete and persistent erection of the penis, which is probably due to a direct or reflex irritation of the nerves of erection. In injuries of the cervical cord particularly, we often see a great and general increase of temperature, up to 110° or 112° F. (43° to 44° C.), especially in severe and rapidly fatal cases; this is of physiological interest, and agrees with the results of experiments. On the other hand, there are also, especially in injuries of the dorsal cord, as it seems, great lowering of temperature, down to 90° or 86° F. (32° to 30° C.).

The further course of the affection differs very much. In the worst cases death ensues in a few hours or days. In other cases the patients recover from the first "shock," but permanent paralysis remains, which may sooner or later lead to death from the ensuing sequelæ, cystitis and bedsores; but we often see partial improvement and eventually a cessation of all the symptoms. Although certain functional disturbances remain permanently, life is not further endangered. Finally, in comparatively mild cases, there may be a complete recovery.

The very characteristic injuries of the conus terminalis and the cauda equina still demand special mention. The conus terminalis is sometimes crushed in fracture of the first lumbar vertebra. Injuries of the conus are produced most frequently by a fall on the buttocks, or even by a fall on the feet with secondary forced flexion of the entire body. The symptoms consist of paralysis of the bladder, rectum, and sexual functions, with anæsthesia of the skin over the sacrum and about the anus, perineum, and genitals (cutaneous branches of the last sacral nerves, compare Fig. 153, page 292). If the injury extend higher there are also sensory disturbances on the posterior aspect of the lower extremities, and paralysis in the region of the sciatic (see Figs. 147 to 152, pages 291 and 292), particularly of the muscles of the lower leg. The paralysis of the bladder is shown by a total loss of all volitional impulses affecting the emptying of this organ. Automatism of the bladder ensues—that is, when the bladder becomes filled up to a certain point it empties itself spontaneously without any effort of the patient. If the tonus of the sphincter be impaired, cystitis and ammoniacal fermentation of the urine with all their sequelæ, are very apt to occur. The rectum empties itself in a similar automatic manner, and frequently unconsciously. The anæsthesia of the buttocks favors the formation of bedsores, which may assume very large dimensions. A similar type of disease to that produced by injuries of the conus, also develops from injury of the cauda equina (for example, from fracture of the lowest end of the vertebral column or fracture of the sacrum), since injury of the root fibers of the sacral and coccygeal plexuses must, of course, cause the same symptoms as injury of the corresponding spinal segments themselves. In such cases we also see the peculiar distribution of the sensory disturbances and the paralyses localized in the sciatic region, especially the double peroneal paralysis that is so very characteristic. The differential diagnosis between an affection of the cauda and a lesion of the lowest portion of the cord is not always easy from the symptoms, but it is of practical importance, especially in regard to the question of any surgical interference. Besides the consideration of the chief point of action of the injury, we must consider especially that symptoms of severe sensory irritation (shooting pains, paræsthesia) favor in general an affection of the cauda, while the existence of dissociated disturbance of sensation is of value in assuming a (central) disease of the spinal cord. The Röntgen rays have caused a very great advance in the matter of estimating correctly all injuries of the vertebræ. With the aid of the Röntgen ray it is now generally possible to learn without difficulty the location and character of injuries of the vertebræ or sacrum.

The *treatment* of the primary affections belongs to the domain of surgery; especially any attempt to lay open the vertebral canal, in order, if possible, to relieve the existing pressure on the cord by reducing the dislocation of the vertebræ or removing the splinters of bone. In most cases, particularly in all cases where the chief injury is to the cord, we have to confine ourselves to putting the patient in a proper position on a water bed, and guarding as carefully as possible against bedsores and cystitis. We can expect but little from local bloodletting, inunctions with mercurial ointment, ice bags, etc. If the first acute stage pass off favorably, the treatment of any paralytic symptoms remaining follows the ordinary rules (baths and electricity). If an extensive bedsore has developed, a cure can often be effected only by means of the permanent water bath.

4. **Diseases of the Spinal Cord after Sudden Reduction of the Atmospheric Pressure** [*Caisson Disease*].—In bridge builders and others who have worked for hours under water in the so-called "caissons," under an external pressure of two or three atmospheres, we sometimes observe peculiar nervous symptoms after leaving the caisson—that is, on the sudden reduction of the atmospheric pressure. These symptoms do not come on immediately after the change to the ordinary atmospheric pressure, but usually some minutes or even half an hour later. They progress very rapidly, so that in a few hours at the latest the disease is fully developed. Besides the frequent mild and transitory symptoms of pain in the ears and hemorrhage from the ears, articular and muscular pains in the back and the extremities, slowing of the pulse and vomiting, vertigo, tinnitus, disturbances of vision and speech, clouding of consciousness, or even complete mental confusion, there are also more persistent and severe disturbances of motility and sensibility, which point unequivocally to an affection of the spinal cord. Usually the spinal symptoms affect the lower extremities chiefly; more rarely they also involve the arms. Paralysis of the diaphragm has also been observed. The type of disease differs much in its details; as a rule, the symptoms of spastic paraplegia predominate, but in other cases there are marked disturbances of sensation or coördination. The bladder is often involved; there is usually retention of urine. The patient sometimes recovers in a few weeks, but in other cases the condition terminates fatally in a comparatively short time—in a few weeks or months. Anatomical investigations (Leyden, F. Schultze, von Schrötter, and others) show in such cases a disseminated but extensive affection in the dorsal cord, chiefly in the posterior columns and the posterior portions of the lateral columns. The nervous tissue in the diseased parts is completely destroyed, and instead of it is found detritus and a collection of large, round, fatty granular cells. Hemorrhages into the cord, which might perhaps be expected, have not been seen. The cause of these small multiple foci of softening is to be found in the occurrence of many gas emboli. During the workman's stay in the compressed air the blood absorbs an abnormal amount of gas, especially nitrogen (Hoppe-Seyler, P. Bert, and others). With too sudden a return to the ordinary conditions of atmospheric pressure, bubbles of gas form in the blood, and, in case they cannot be thrown off fast enough by the lungs, they lead to numerous emboli in the smaller vessels with their consequences. The anatomical arrangement of the vessels seems to be the reason why the embolic foci are situated chiefly in the dorsal cord.

The treatment is the same as in acute myelitis. The prophylactic measures to be employed in a given case are, of course, very important. The onset of morbid symptoms can be certainly avoided by the cautious and slow reduction of the atmospheric pressure. [No person suffering from any disease, especially of the heart or kidneys, no one addicted to alcohol, and no old or very stout person, should enter a caisson. It is probably safer not to enter a caisson fasting. Beginners should remain in the caisson a comparatively short time—not over an hour. For more experienced workmen a day's work should not exceed four hours, divided into two shifts with four hours' interval. Great precautions must also be taken against cold. The process of "unlocking" must be slow, at least five minutes for each atmosphere of pressure. It is said that when the symptoms first come on they may be averted by restoring the victim to the caisson or to a cabinet capable of affording increased atmospheric pressure. Fluid extract of ergot (1 drachm [gm. 4] every hour for a few doses) is said to give great relief at the outset.—K.]

CHAPTER IV

THE PRESSURE PARALYSES OF THE SPINAL CORD

(Slow Compression of the Spinal Cord, especially in Caries and Carcinoma of the Vertebræ)

Ætiology.—Many pathological processes in the vicinity of the spinal cord exert a gradually increasing pressure upon it, and thus inhibit the conduction of nervous irritation, and even cause coarse organic changes in the substance of the cord. The seat of such affections is, in the first place, in the membranes of the spinal cord. In the chapter on Meningitis we have already mentioned the compressing action of the masses of inflammatory exudation on the nerve roots and the cord, and we have learned to recognize, especially in pachymeningitis cervicalis hypertrophica, a characteristic example of a gradually increasing compression of the cervical cord. Precisely similar conditions are found in the not very rare meningeal tumors, whose special pathology will be described in connection with the tumors of the cord itself.

By far the most frequent pressure paralyses of the spinal cord, and hence the most important practically, are caused by certain diseases of the vertebræ, and, first of all, by tuberculosis of the vertebræ, the so-called caries of the vertebræ (spondylitis, Pott's disease, spondylarthrocace).[1] Tubercular spondylitis occurs at almost any age; it is rare only in old people. It often develops in children, but it is almost as frequent in adults. The ætiological significance of injuries, such as a fall or a blow, which are often mentioned by the patients themselves or their parents, is in most cases doubtful, but not wholly to be ignored. As a rule, vertebral caries apparently develops quite independently as a primary disease, but we can very often succeed in finding ætiological factors rendering tuberculous disease likely—the tuberculous habit, hereditary tendency, or previous tuberculous disease elsewhere, such as phthisis, pleurisy, other affections of the bones, etc.

Cancer of the vertebræ, as well as caries, leads to pressure paralysis of the spinal cord, but it is relatively much rarer than caries; it develops chiefly in older persons, and it occurs only as a secondary new growth in cancer of other organs—the breast, the stomach, the œsophagus, etc. Sarcoma is the chief primary tumor of the vertebræ, but it may also involve the vertebræ secondarily by extension from growths in the vicinity.

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[1] In the kyphoscoliosis of the vertebral column, not due to spondylitis, but caused by abnormal mechanical conditions or conditions of growth, there are practically never any symptoms of compression in the cord, even in very pronounced cases. In these cases the cord manifestly shows quite a great adaptability.

We must mention here briefly, as very rare causes of compression of the spinal cord, aneurism of the aorta, which gradually erodes the vertebræ, echinococci in the vertebral canal, exostoses of the vertebræ, [myeloma], and syphilitic new growths.

Pathological Anatomy.—Vertebral caries is by far most frequent in the dorsal portion of the vertebral column (dorsal spondylitis), less frequent in the cervical portion (cervical spondylitis), and rarest in the lumbar portion (lumbar spondylitis) and in the sacrum (sacral spondylitis). It usually extends over several adjacent vertebræ, or more rarely two separate foci of disease are seen. The process itself, the details of which cannot be discussed here, probably always begins in the spongy substances of the bodies of the vertebræ. We see here, on section, in incipient cases, roundish, pale reddish, or yellowish nodules, which consist of newly formed fungous tissue—that is, tubercular granulation tissue. The bony substance becomes more and more destroyed by the invasion of the new growth, which itself shows the characteristic tendency of all tubercular new growth to cheesy degeneration. Thus there is often an extensive destruction of the bodies of the vertebræ, which later on involves the vertebral processes also, the intervertebral discs, and the other articular connections between the different vertebræ.

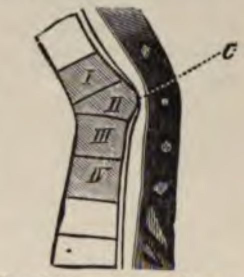

FIG. 155.—Schematic representation of vertebral displacement in spondylitis. The point of compression of the cord, at the level of the second dorsal vertebra, is at *C*.

There are essentially two factors to be considered in regard to the question which chiefly interests us here—that is, in regard to the occurrence of compression of the spinal cord. In the first place, it is clear that the complete or partial destruction of the bodies of one or even more vertebræ, and of their articular connections, cannot remain without influence upon the position of the other adjacent vertebræ. In fact, we very often see dislocations of the vertebræ as a result of it, usually by the pushing backward of the partly destroyed vertebra by the movements of the vertebræ above and below the diseased portion on one another (see Fig. 155). There arises, on the one hand, a contraction of the vertebral canal, and with it often a very considerable limitation of the space for the cord; and, on the other hand, that characteristic projection of the spinous processes in the region of the diseased portion of the vertebral column which forms the so-called Pott's boss—the angular kyphosis (Fig. 156, page 304). In very slight degrees of the disease there is only a trifling projection of one or more spinous processes, but in other cases it gradually becomes an extensive deformity of the vertebral column, which strikes us at the first glance; but it must be especially borne in mind that in very many cases of vertebral caries, where the body of no vertebra is wholly or mainly destroyed, there is no knuckle formed at all. The line of the spinous processes in such cases preserves completely its normal external form.

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[illegible] of the cord [illegible] are affected, the
[illegible] diminished; the
[illegible] old cases [illegible] find the cord
[illegible] (*vide infra*). It is very remarkable,

however, that often, as we have repeatedly seen, marked symptoms of paralysis may be present during life without finding any coarse mechanical lesion of the cord in the cadaver, so that the cord may have an almost perfectly normal appearance. As in the peripheral nerves, so in the spinal cord, a moderate pressure is manifestly enough to cause a partial break in the conduction, without being at the same time necessarily associated with an actual mechanical destruction of nervous elements. On careful microscopic examination of the cord, we find in such cases, in spite of the existence of a complete paraplegia during life, that most of the nerve fibers are still completely preserved, and that there are only here and there a few lacunæ, corresponding to circumscribed lymph-spaces (*vide supra*), and to single fibers which have been destroyed. This discovery is especially interesting because it makes us understand the possibility of recovery, even in apparently severe cases (*vide infra*).

If we take a portion from the soft place of compression for examination in the fresh state, we find usually many, sometimes (in old cases) only a few, granular cells, according to the amount of disintegrated nerve medulla, the remains of which are taken up by the white blood corpuscles (the wandering cells), and probably also by the glia cells. If we make stained cross sections of the hardened cord, we see under the microscope no signs of hyperæmia, or accumulation of cells about the vessels, and only exceptionally a little hemorrhage; but we do find, besides many still preserved nerve fibers, other fibers, which are involved in the disintegration or are already destroyed. Very commonly the changes are distributed in the form of nodules. We find groups of greatly swollen axis cylinders, or of axis cylinders torn off and rolled up in a spiral. The medullary sheaths are everywhere involved in the disintegration, until they blend with the disintegrated axis cylinders. The products of disintegration developing everywhere serve, as was said above, to form the granular cells. The ganglion cells and the glia also show signs of disintegration. We not infrequently find the so-called corpora amylacea, whose origin is still somewhat obscure. If the destruction of the nervous tissue have advanced to a certain degree, there is in the later stages, as in all analogous processes, a secondary implication of the neuroglia. Now follows an increase of the interstitial connective tissue. Its proliferations, which take the place of the destroyed nervous tissue, seem broad and thick—at first loose, but later firm and fibrillary. Thus it happens that in old cases we find nothing at the point of compression but a loss of nerve fibers in the cord, and instead a firm fibrous tissue. We can sometimes notice processes of regeneration in the nerve fibers (*vide infra*). In general the changes are much more marked in the white matter of the cord than in the gray.

Finally, after every protracted compression of the cord we find an ascending and descending secondary degeneration of certain systems of fibers in the cord (*vide infra*).

We need not go more fully into the details of compression of the cord from other causes, since the results, as far as they are of a purely mechanical nature, are precisely the same. In cancer of the vertebræ there may also be dislocations of the vertebral column after the destruction of some of the vertebræ, but usually the compression depends upon the direct growth of the newly formed tissue into the dura. In these cases the compression of the nerve roots in the intervertebral foramina is also of importance.

Clinical History.—1. THE PRESSURE PARALYSES IN VERTEBRAL CARIES.—Many cases of spondylitis run their course without involving the cord, or at least they involve it only in quite a subordinate fashion. In other cases the symptoms of disease of the vertebræ exist for a long time alone, until at last, slowly or suddenly, the signs of compression of the cord are added to them. Finally, in a third group of cases, the vertebral disease is so latent that only the cord symptoms are prominent in the type of the disease, and the disease of the vertebræ is easily entirely overlooked.

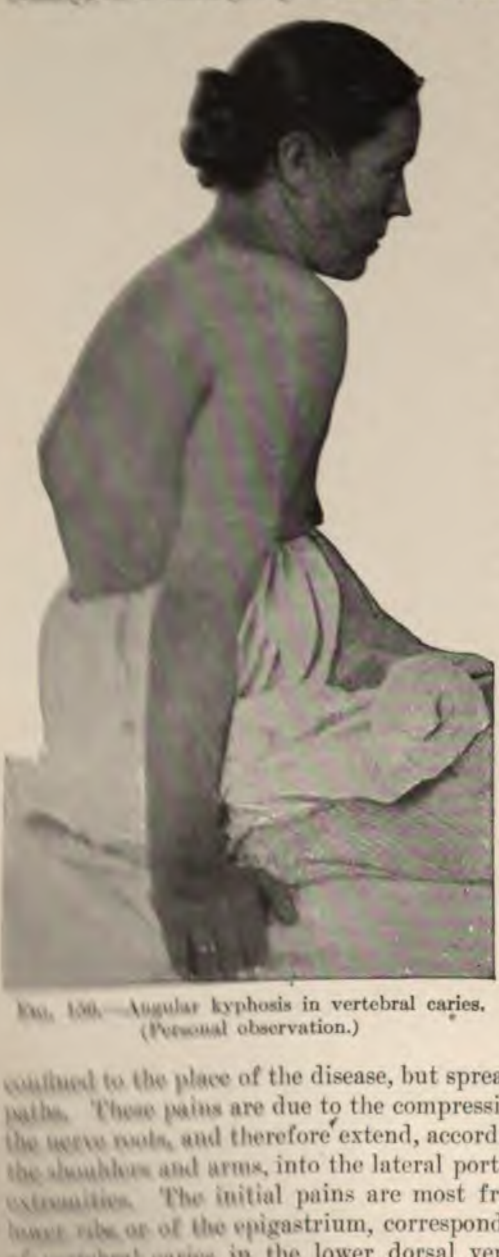

FIG. 156.—Angular kyphosis in vertebral caries. (Personal observation.)

Usually the symptoms of the developing primary disease, the affection of the vertebræ, precede the appearance of the first cord symptoms by some time. The patient feels a dull pain at a definite part of the spine, which is increased by movements of the trunk, by bending or straightening up. Many patients notice the stiffness of the vertebral column of themselves, and sometimes even a beginning deformity. In characteristic cases we notice at once, on examining the vertebral column, an angular kyphosis (gibbus, Fig. 156) of considerable extent or limited to a few vertebræ; but sometimes there is only a slight prominence of a single vertebra, and not infrequently there is externally no change in the formation of the vertebral column. The tenderness on pressure on the spinous processes is also not a constant symptom; it may be very pronounced, but it is not infrequently absent even in marked kyphosis. The first cord symptoms usually consist of painful sensations, which are not confined to the place of the disease, but spread out along the course of the nerve paths. These pains are due to the compression and the consequent irritation of the nerve roots, and therefore extend, according to the seat of the affection, into the shoulders and arms, into the lateral portions of the trunk, or into the lower extremities. The initial pains are most frequently felt in the region of the lower ribs or of the epigastrium, corresponding to the most frequent situation of vertebral caries in the lower dorsal vertebræ. They are sometimes very severe, and then they usually have a pronounced neuralgiform character, or they may be more dull, pressing, dragging, etc. Besides the special pains, there are also many forms of paræsthesia, such as formication, cold feelings, and burning feelings.

The disturbances of motility begin to appear at the same time with these symptoms or soon after them. A stiffness and weakness arise which impede the gait more and more, usually not in both legs at once, but first in one and then in the other, although, as a rule, both legs are soon involved. This paresis increases rapidly or slowly, and it may finally go on to a complete motor paralysis. If the seat of the affection be in the dorsal vertebræ, as it usually is, or if it be in the lumbar vertebræ, the paralysis affects the lower extremities only, and the arms, of course, remain intact; but in cervical spondylitis the arms are usually first and chiefly affected. Only on great compression of the cervical cord is the conduction of the fibers passing through it for the lower extremities impaired, and then there are also disturbances of function in these. Ataxia of the legs is not very common, but it certainly does occur in compression of the cord. The ataxia, of course, is usually concealed by the coexisting paresis of the muscles or the existing spastic conditions. We see ataxia most frequently in the stages of development of, or recovery from, compression paralyses.

Disturbances of sensibility are quite often found, independently of the pains and paræsthesiæ above mentioned, but in many cases of pressure paralysis they are present in only a comparatively slight degree. It seems that the sensory nerves resist pressure more than the motor nerves, just as in the pressure paralyses of peripheral nerves; but possibly their position in the gray matter of the posterior cornua protects them better from the above-mentioned mechanical injuries than is the case, for example, with the motor fibers in the pyramidal tract. The fact is that often, even in complete motor paraplegia, there is little, if any, diminution of sensibility, and that marked anæsthesia is rare, and is seen only in the last stages of the disease. We find most frequently a slight blunting of sensibility equally to all forms of sensation, especially to the sense of pain; in other cases partial disturbances of the sense of temperature with the sensibility to touch and pain well preserved, sometimes partial anæsthesia of the sense of pressure, etc. The different parts of the skin not infrequently act differently, so that we find portions with quite normal sensibility, as well as very anæsthetic portions. There may also be hyperæsthetic areas of the skin (especially at the upper boundary of the disturbance of sensation), particularly with marked hyperæsthesia to pain (pin pricks).

The condition of the reflexes is interesting. If the seat of the compression be above the reflex arc, which we must assume to be in the lumbar cord for the reflexes of the lower extremities, we should expect that the reflexes would persist, and in many cases even be increased, corresponding to the decline in the inhibitory influences coming from above. The latter takes place in most cases with the tendon reflexes. The increase of the tendon reflexes, and the coincident increase of muscle tonus, may reach so great a degree in many cases of compression of the upper dorsal or cervical cord as to show in the lower extremities the pronounced type of spastic rigidity and spastic paralysis. The legs are then found in rigid tonic extension, with the knees pressed firmly together, or there is spastic contracture of the flexors and adductors. If the patient can still flex his leg, each effort is attended in many cases by a marked protrusion of the tendon of the tibialis anticus muscle ("tibialis phenomenon"). All passive movements of the legs can be performed only with the greatest difficulty, and the attempt often causes a general tremor of the leg. All the tendon reflexes (patellar reflex, adductor reflex, Achilles reflex, etc.) are much increased, but we must remember that even in apparently flaccid paraplegia the reflexes are sometimes quite lively. Spastic paraplegia is produced only by an increase of muscular tonus (hypertonia). Muscular tonus and the tendon reflexes are not, however, always affected in a similar way. In cervical spondylitis the tendon and periosteal reflexes in the arms are also increased sometimes, but in other cases, if the reflex arc be injured, they are absent. Where the seat of the compression is above the lumbar cord, the cutaneous reflexes show frequently also considerable vigor. The plantar reflex of the big toe occurs as a pronounced dorsal extension (Babinski's reflex, [Oppenheim's reflex], *vide supra*, page 208), if spastic paralysis be present. In some severe pressure paralyses in the dorsal cord the cutaneous reflexes are diminished. They are rarely entirely absent, but one must understand testing them, and must employ long-continued cutaneous irritation, such as pinching and pricking, in different parts of the skin in order to provoke them (*vide supra*, page 208). In cases of total compression of the cervical cord we sometimes find a flaccid paraplegia with absence of the tendon reflexes in the legs, just as in traumatic total transverse lesion of the cervical cord. There has been much discussion as to the explanation of this remarkable phenomenon. In all such cases there is probably a secondary change in the lumbar cord, by which the reflex arc is directly interfered with (*vide supra*, page 297).

Disturbances of the rectum and bladder occur in almost all severe cases of pressure paralysis. The difficulty of micturition is often an early symptom of the disease; later on there is complete retention of urine, and, in the more advanced stages of the disease, there is usually incontinence—that is, a reflex voiding of urine. With this the danger of the development of cystitis becomes very great. The bowels are usually constipated, but sometimes there is also incontinence of fæces.

Trophic disturbances are often found in the paralyzed parts. If there be severe symptoms of sensory irritation there may sometimes be eruptions of herpes, corresponding to the course of the nerves. More frequently there are chronic disturbances in the nutrition of the skin in severe and long-continued cases. It becomes dry, the epidermis scales off, and the nails become brittle. Bedsores form very easily in severe cases on the sacrum, on the buttocks, on the inner side of the knees, and on the heels, especially when the patient has insufficient care. The paralyzed muscles soon atrophy to a greater or less extent. The normal electrical reaction is preserved, however, if the corresponding spinal motor centers are unaffected. If the motor ganglion cells of the lumbar segment be destroyed, reaction of degeneration ensues in the corresponding muscles of the leg. In cervical spondylitis with lesion of the cervical cord or of the anterior cervical roots there may be atrophic paralysis in the arms. We have already learned to recognize the peculiar type of nervous symptoms in disease of the lowest portion of the cord or of the cauda equina (page 298). We sometimes see the same type in caries of the upper lumbar vertebræ with compression of the conus, or in caries of the sacrum with compression of the cauda equina. The points to be noticed in distinguishing between affections of the conus and affections of the cauda have already been mentioned on page 298.

Thus we see, under some circumstances, in compression of the cord, the whole group of symptoms arising which are the necessary consequence of the break in conduction in the spinal cord, and which we shall likewise meet again in various other spinal affections, especially in myelitis and in tumors. The intensity and number of the symptoms must, of course, vary very much in the different cases. If the compression be quite slight,. there are only mild symptoms of sensory irritation and slight paresis. One of the earliest and most constant signs of a compression of the cord, in the dorsal or cervical region, is a decided increase of the patellar reflex. We sometimes find it at a time when there is scarcely a single other cord symptom present. If the compression increases, the paresis becomes more marked, the disturbance of sensibility is greater, and vesical disturbances arise, until finally the complete type of an entire transverse interruption of conduction in the cord is developed. The precise limitation of the symptoms, especially the distribution of the sensory disturbance, follows the rules already given (page 289). There is only rarely, however, a complete transverse destruction of the spinal cord in vertebral caries. The conduction of sensory impressions, at least, is usually not wholly abolished. The time required for the development of the symptoms of spinal compression differs very much. They sometimes attain a considerable height in a short time, and sometimes they develop only after a course of months. Variations in the intensity of the symptoms are frequent, and they point perhaps to a corresponding variation in the severity of the compression.

In regard to the result of pressure paralyses in vertebral caries, spondylitic processes without doubt can recover, which is by no means in contradiction to their character as a local tubercular process. In this connection the fact is of great practical importance that even the pressure paralyses may be completely restored, as far as the cause of the compression can be removed by the absorption of inflammatory and tuberculous new growths, so that a complete and permanent recovery may take place even after the paralysis has lasted for some months, or a year, or even a year and a half. Such recoveries have been seen in a considerable number of cases by others and also by ourselves. They are explained in part by the restoration of function in fibers which were rendered merely incapable of conduction by pressure, but not completely destroyed (page 303), and in part perhaps in old cases by the occurrence of regeneration. Fickler has made the interesting observation in my clinic that the ends of the pyramidal fibers in the lateral tract may grow out above an old compression, pass around the scar of the compression through the anterior fissure, and enter the cord below it to their anterior horns.

Although, according to this, the prognosis in a part of the cases of pressure paralysis from spondylitis is comparatively good, still, many other cases terminate unfavorably. The cause lies either in the occurrence of dangerous sequelæ of the paralysis, such as bedsores, cystitis, or pyelonephritis, with fever and increasing general weakness; or in the development of other tubercular diseases, especially phthisis, or more rarely miliary tuberculosis or tubercular meningitis, which prove fatal. In some cases there is a stationary

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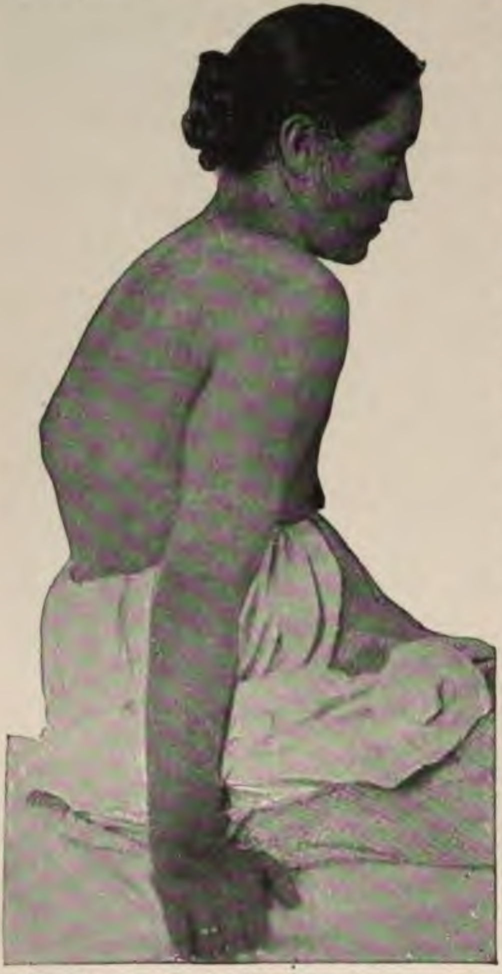

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Usually the symptoms of the developing primary disease, the affection of the vertebræ, precede the appearance of the first cord symptoms by some time. The patient feels a dull pain at a definite part of the spine, which is increased by movements of the trunk, by bending or straightening up. Many patients notice the stiffness of the vertebral column of themselves, and sometimes even a beginning deformity. In characteristic cases we notice at once, on examining the vertebral column, an angular kyphosis (gibbus, Fig. 156) of considerable extent or limited to a few vertebræ; but sometimes there is only a slight prominence of a single vertebra, and not infrequently there is externally no change in the formation of the vertebral column. The tenderness on pressure on the spinous processes is also not a constant symptom; it may be very pronounced, but it is not infrequently absent even in marked kyphosis. The first cord symptoms usually consist of painful sensations, which are not confined to the place of the disease, but spread out along the course of the nerve paths. These pains are due to the compression and the consequent irritation of the nerve roots, and therefore extend, according to the seat of the affection, into the shoulders and arms, into the lateral portions of the trunk, or into the lower extremities. The initial pains are most frequently felt in the region of the lower ribs or of the epigastrium, corresponding to the most frequent situation of vertebral caries in the lower dorsal vertebræ. They are sometimes very severe, and then they usually have a pronounced neuralgiform character, or they may be more dull, pressing, dragging, etc. Besides the special pains, there are also many forms of paræsthesia, such as formication, cold feelings, and burning feelings.

The disturbances of motility begin to appear at the same time with these symptoms or soon after them. A stiffness and weakness arise which impede the gait more and more, usually not in both legs at once, but first in one and then in the other, although, as a rule, both legs are soon involved. This paresis increases rapidly or slowly, and it may finally go on to a complete motor paralysis. If the seat of the affection be in the dorsal vertebræ, as it usually is, or if it be in the lumbar vertebræ, the paralysis affects the lower extremities only, and the arms, of course, remain intact; but in cervical spondylitis the arms are usually first and chiefly affected. Only on great compression of the cervical cord is the conduction of the fibers passing through it for the lower extremities impaired, and then there are also disturbances of function in these. Ataxia of the legs is not very common, but it certainly does occur in compression of the cord. The ataxia, of course, is usually concealed by the coexisting paresis of the muscles or the existing spastic conditions. We see ataxia most frequently in the stages of development of, or recovery from, compression paralyses.

Disturbances of sensibility are quite often found, independently of the pains and paræsthesiæ above mentioned, but in many cases of pressure paralysis they are present in only a comparatively slight degree. It seems that the sensory nerves resist pressure more than the motor nerves, just as in the pressure paralyses of peripheral nerves; but possibly their position in the gray matter of the posterior cornua protects them better from the above-mentioned mechanical injuries than is the case, for example, with the motor fibers in the pyramidal tract. The fact is that often, even in complete motor paraplegia, there is little, if any, diminution of sensibility, and that marked anæsthesia is rare, and is seen only in the last stages of the disease. We find most frequently a slight blunting of sensibility equally to all forms of sensation, especially to the sense of pain; in other cases partial disturbances of the sense of temperature with the sensibility to touch and pain well preserved, sometimes partial anæsthesia of the sense of pressure, etc. The different parts of the skin not infrequently act differently, so that we find portions with quite normal sensibility, as well as very anæsthetic portions. There may also be hyperæsthetic areas of the skin (especially at the upper boundary of the disturbance of sensation), particularly with marked hyperæsthesia to pain (pin pricks).

The condition of the reflexes is interesting. If the seat of the compression be above the reflex arc, which we must assume to be in the lumbar cord for the reflexes of the lower extremities, we should expect that the reflexes would persist, and in many cases even be increased, corresponding to the decline in the inhibitory influences coming from above. The latter takes place in most cases with the tendon reflexes. The increase of the tendon reflexes, and the coincident increase of muscle tonus, may reach so great a degree in many cases of compression of the upper dorsal or cervical cord as to show in the lower extremities the pronounced type of spastic rigidity and spastic paralysis. The legs are then found in rigid tonic extension, with the knees pressed firmly together, or there is spastic contracture of the flexors and adductors. If the patient can still flex his leg, each effort is attended in many cases by a marked protrusion of the tendon of the tibialis anticus muscle ("tibialis phenomenon"). All passive movements of the legs can be performed only with the greatest difficulty, and the attempt often causes a general tremor of the leg. All the tendon reflexes (patellar reflex, adductor reflex, Achilles reflex, etc.) are much increased, but we must remember that even in apparently flaccid paraplegia the reflexes are sometimes quite lively. Spastic paraplegia is produced only by an increase of muscular tonus (hypertonia). Muscular tonus and the tendon reflexes are not, however, always affected in a similar way. In cervical spondylitis the tendon and periosteal reflexes in the arms are also increased sometimes, but in other cases, if the reflex arc be injured, they are absent. Where the seat of the compression is above the lumbar cord, the cutaneous reflexes show frequently also considerable vigor. The plantar reflex of the big toe occurs as a pronounced dorsal extension (Babinski's reflex, [Oppenheim's reflex], *vide supra,* page 208), if spastic paralysis be present. In some severe pressure paralyses in the dorsal cord the cutaneous reflexes are diminished. They are rarely entirely absent, but one must understand testing them, and must employ long-continued cutaneous irritation, such as pinching and pricking, in different parts of the skin in order to provoke them (*vide supra,* page 208). In cases of total compression of the cervical cord we sometimes find a flaccid paraplegia with absence of the tendon reflexes in the legs, just as in traumatic total transverse lesion of the cervical cord. There has been much discussion as to the explanation of this remarkable phenomenon. In all such cases there is probably a secondary change in the lumbar cord, by which the reflex arc is directly interfered with (*vide supra*, page 297).

Disturbances of the rectum and bladder occur in almost all severe cases of pressure paralysis. The difficulty of micturition is often an early symptom of the disease; later on there is complete retention of urine, and, in the more advanced stages of the disease, there is usually incontinence—that is, a reflex voiding of urine. With this the danger of the development of cystitis becomes very great. The bowels are usually constipated, but sometimes there is also incontinence of fæces.

Trophic disturbances are often found in the paralyzed parts. If there be severe symptoms of sensory irritation there may sometimes be eruptions of herpes, corresponding to the course of the nerves. More frequently there are chronic disturbances in the nutrition of the skin in severe and long-continued cases. It becomes dry, the epidermis scales off, and the nails become brittle. Bedsores form very easily in severe cases on the sacrum, on the buttocks, on the inner side of the knees, and on the heels, especially when the patient has insufficient care. The paralyzed muscles soon atrophy to a greater or less extent. The normal electrical reaction is preserved, however, if the corresponding spinal motor centers are unaffected. If the motor ganglion cells of the lumbar segment be destroyed, reaction of degeneration ensues in the corresponding muscles of the leg. In cervical spondylitis with lesion of the cervical cord or of the anterior cervical roots there may be atrophic paralysis in the arms. We have already learned to recognize the peculiar type of nervous symptoms in disease of the lowest portion of the cord or of the cauda equina (page 298). We sometimes see the same type in caries of the upper lumbar vertebræ with compression of the conus, or in caries of the sacrum with compression of the cauda equina. The points to be noticed in distinguishing between affections of the conus and affections of the cauda have already been mentioned on page 298.

Thus we see, under some circumstances, in compression of the cord, the whole group of symptoms arising which are the necessary consequence of the break in conduction in the spinal cord, and which we shall likewise meet again in various other spinal affections, especially in myelitis and in tumors. The intensity and number of the symptoms must, of course, vary very much in the different cases. If the compression be quite slight, there are only mild symptoms of sensory irritation and slight paresis. One of the earliest and most constant signs of a compression of the cord, in the dorsal or cervical region, is a decided increase of the patellar reflex. We sometimes find it at a time when there is scarcely a single other cord symptom present. If the compression increases, the paresis becomes more marked, the disturbance of sensibility is greater, and vesical disturbances arise, until finally the complete type of an entire transverse interruption of conduction in the cord is developed. The precise limitation of the symptoms, especially the distribution of the sensory disturbance, follows the rules already given (page 289). There is only rarely, however, a complete transverse destruction of the spinal cord in vertebral caries. The conduction of sensory impressions, at least, is usually not wholly abolished. The time required for the development of the symptoms of spinal compression differs very much. They sometimes attain a considerable height in a short time, and sometimes they develop only after a course of months. Variations in the intensity of the symptoms are frequent, and they point perhaps to a corresponding variation in the severity of the compression.

In regard to the result of pressure paralyses in vertebral caries, spondylitic processes without doubt can recover, which is by no means in contradiction to their character as a local tubercular process. In this connection the fact is of great practical importance that even the pressure paralyses may be completely restored, as far as the cause of the compression can be removed by the absorption of inflammatory and tuberculous new growths, so that a complete and permanent recovery may take place even after the paralysis has lasted for some months, or a year, or even a year and a half. Such recoveries have been seen in a considerable number of cases by others and also by ourselves. They are explained in part by the restoration of function in fibers which were rendered merely incapable of conduction by pressure, but not completely destroyed (page 303), and in part perhaps in old cases by the occurrence of regeneration. Fickler has made the interesting observation in my clinic that the ends of the pyramidal fibers in the lateral tract may grow out above an old compression, pass around the scar of the compression through the anterior fissure, and enter the cord below it to their anterior horns.

Although, according to this, the prognosis in a part of the cases of pressure paralysis from spondylitis is comparatively good, still, many other cases terminate unfavorably. The cause lies either in the occurrence of dangerous sequelæ of the paralysis, such as bedsores, cystitis, or pyelonephritis, with fever and increasing general weakness; or in the development of other tubercular diseases, especially phthisis, or more rarely miliary tuberculosis or tubercular meningitis, which prove fatal. In some cases there is a stationary paralytic condition, since the vertebral disease recovers and sclerosis ensues in the cord, which is capable of no further change.

2. THE PRESSURE PARALYSES IN CANCER OF THE VERTEBRÆ.—In pressure paralysis from cancer of the vertebræ, which is not very rare, the spinal affection may seem to be primary, especially when the primary growth (breast, etc.) has not up to that time caused any very striking clinical symptoms. The first symptom is usually pain, both in the region of the new growth and radiating in neuralgic form. The pain becomes distressing if the tumor grows about and compresses the posterior roots (*"paraplegia dolorosa"*). The other spinal symptoms are precisely analogous to those in compression of the cord by vertebral caries, and therefore they need not be described again in detail. Changes in the shape of the vertebral column sometimes occur, but we only rarely find a characteristic angular gibbus, as in vertebral tuberculosis. The general cancerous cachexia is usually soon apparent. The course is in general more rapid than in caries. The disease rarely lasts more than nine to eighteen months before its invariably fatal end.

Diagnosis.—The frequency of the pressure paralyses of the spinal cord admonishes us to examine the vertebral column carefully in every case of spinal disease, especially if the case in question cannot be referred to one of the special types of systemic diseases (*vide infra*). We should look especially for the stiffness of special parts of the vertebral column on movements of the head or trunk, and also for the pronounced tenderness of single vertebræ to pressure, and, finally, as a most important and most certain sign, for the deformity of the vertebral column, the marked projection of a single spinous process, or the formation of an evident angular kyphosis. If we find such a Pott's boss the diagnosis is easy, and we can then at any rate refer the existing cord symptoms to a compression of the cord caused by a disease of the vertebræ.

The diagnosis is more difficult if the signs of an affection of the vertebræ be not evident. It must once for all be stated that vertebral caries does not invariably and necessarily result in a manifest knuckle, and that even the tenderness of the vertebræ to pressure is sometimes very slight in spondylitis. In such cases the examination of the vertebral column must be repeated frequently, since even slight anomalies obtain a diagnostic value if constantly present. The Röntgen-ray examination of the spinal column is of the greatest diagnostic importance. In good Röntgen photographs the diseased vertebræ can generally be readily recognized (differences in shape, changes in the intervertebral discs, changes in the structure of the vertebræ, etc.).

Clinically, the most characteristic features of a compression of the cord are its beginning with symptoms of sensory irritation, the persistent and more or less severe pain in the back, the preponderance of symptoms of motor paralysis with comparatively little disturbance of sensibility, and finally the frequent asymmetry of the symptoms on the two sides, which may even recall the type of the so-called "unilateral lesion" of the spinal cord (*vide infra*). Sometimes the cause of the spinal symptoms is at first obscure, and later on in the disease a marked anomaly of the vertebral column develops.

If the diagnosis of an affection of the vertebræ be certain, the next question is as to the nature of it, especially whether we have to do with a spondylitis or with a cancer of the vertebræ. Since spondylitis is by far the more frequent disease, we must always think of that first, especially in young people predisposed to tuberculosis, and where we have the formation of a pronounced angular kyphosis. Temperature is also important in the diagnosis of caries of the vertebræ. Slight elevations of temperature, which are so characteristic of all tuberculous diseases, frequently help to establish the diagnosis of spondylitis. Marked increase in the frequency of the pulse, when the temperature is approximately normal, to a certain extent also favors the assumption of a tuberculous spondylitis. In cancer of the vertebræ the coarser changes in the form of the vertebral column are generally less marked. This affection develops usually in older people, after the age of forty, and is manifested, as we have said, by the great intensity of the initial symptoms of sensory irritation. The discovery of a primary cancer is necessary in the first place. Thus, for example, cancer of the vertebræ occurs at times secondarily to a primary carcinoma of the stomach. By far the greater number of cases of cancer of the vertebræ which I have seen occurred secondarily to cancer of the breast. In some cases the primary tumor had been extirpated, and, nevertheless (not infrequently even without any demonstrable local recurrence), a secondary carcinoma of the spine had developed. If patients with carcinoma of the breast (operated on or not) suffer from continuous pain in the back and lumbar region, we may generally assume that the onset of this sad complication has occurred. The presence of carcinomatous lymph-glands in the inguinal region may occasionally assist in making a diagnosis. Finally, a certain stress is to be laid on the well-known general habit of patients with cancer, on the peculiar cancerous cachexia, and on the whole course of the disease.

In regard to the distinction between caries of the vertebræ and sarcoma of the dura mater, see the chapter on Tumors of the Spinal Cord.

The place of compression is, in the majority of cases, to be recognized by the evident localization of the disease of the vertebræ. The Röntgen-ray examination generally gives us information that is of the greatest value. In other respects the same rules hold for localization which we have given in detail in the second chapter of this section (page 285 *et seq.*).

Treatment.—In regard to the special treatment of spondylitis, especially the orthopedic treatment, we must refer to the text-books of surgery. The most benefit is to be expected from the various contrivances for extension of the spinal column. It cannot be denied, and we have ourselves several times seen, that such extension sometimes produces a decided diminution of the pressure on the cord, which is followed by a remarkably rapid improvement of certain symptoms of paralysis. Such observations are strongly in favor of our theory that the action of vertebral caries is usually purely mechanical. Very often, however, we have had to admit that the extension apparatus was of no benefit or only of temporary benefit, or that it even materially aggravated the patient's distress. In vertebral caries with coexisting spinal symptoms we must therefore use extension of the vertebræ with caution. The use of mechanical supports and jackets (Sayre's plaster jacket, Lorenz's plaster bed, etc.), which are also recommended, cannot be considered here in detail. Surgical interference (laminectomy) to remove compressing masses of pus, etc., has been repeatedly tried in vertebral caries, sometimes with benefit, but often without success. At any rate, we should decide on operation only after other methods of treatment have been unsuccessful.

In most cases we are limited to a general symptomatic and dietetic treatment. Persistent rest in bed, on the back, is of the greatest importance, and it must always be strongly advised. Several cases of complete recovery from severe pressure paraplegia, which we have had under our own observation, were due to this alone. The most careful treatment of the general condition (fresh air, good food, cod-liver oil, arsenic, iron, etc.) of course also demands attention. We have seen very unfavorable action from Koch's tuberculin. The reaction of the tuberculous focus may be followed by an increase in the symptoms of compression. Local applications to the vertebral column are much used, particularly green soap, painting with iodin, and especially the actual cautery. The use of the latter in spondylitis has even to-day warm advocates, and, in fact, deserves to be tried, the procedure with Paquelin's thermocautery being especially easy; we make some three or four eschars on each side of the diseased vertebræ.

Among other remedies we may mention stabile galvanization at the point of pressure, and the electrical treatment of the paralyzed extremities; also the use of baths, especially salt baths. In regard to the symptomatic treatment, we will refer to the following chapter.

CHAPTER V

ACUTE AND CHRONIC MYELITIS

(Transverse Myelitis. Diffuse Myelitis)

Definition and Ætiology of Myelitis.—"Myelitis" is the name we give to those inflammatory degenerative diseases of the spinal cord which develop primarily in the substance of the cord itself and lead to a more or less extensive destruction of the nervous elements. We know very little of the causes of these diseases and of the precise nature and manner in which these causes exert their morbid influence. The name of myelitis has been used until recently for several distinct and totally dissimilar morbid processes, since it is often by no means easy to recognize the precise origin of the anatomical changes. At any rate, we must recognize this uncertainty in the definition of myelitis, and at least try as much as possible to keep separate the forms which surely do not belong in the same group. Thus we have said expressly in the previous chapter that the purely mechanical traumatic changes in the spinal cord are to be differentiated from true myelitis and that the inflammatory processes in these cases are at most secondary. We must also emphasize the fact that anæmic necrosis of the tissues from vascular obstruction (from primary endarteritis, thrombosis, or embolism of the vessels) does not seem to have the significance in the cord that it does in the brain, but that we must always bear in mind the possibility of its occurrence, and hold firmly to the fundamental distinction between such anæmic softening or infarction and primary degenerative inflammation. It is very difficult to determine the position which syphilis holds in relation to the definition of myelitis. The distinction is, of course, easy in the rare cases where there is an actual gumma in the cord, but there are also a good many cases of apparently genuine degenerative myelitis in persons who have previously had syphilis. In these cases the anatomical findings often leave us in the lurch, if the question of the syphilitic origin of the disease is to be answered with certainty. Even in cases where this origin is recognized, the way in which the syphilitic agent acts is still utterly obscure. Most stress is generally laid, of course, upon the "specific syphilitic" changes in the blood vessels; but these criteria are not wholly unequivocal in their nature, and the question of the mutual relations between the disease of the vessels and the true parenchymatous changes still needs further discussion. It is, however, of practical importance to know that syphilis apparently plays a considerable part in the ætiology of the spinal diseases hitherto classed as myelitis. Myelitis of the upper dorsal cord, which runs its course chiefly under the form of spastic spinal paralysis (*q. v.*), seems frequently to be due to previous syphilis. We will return to this question in the chapter on Syphilis of the Spinal Cord. The recent sero-diagnostic methods of examination will also be discussed under the same heading.

We can call only those processes genuine myelitis in which some excitant of inflammation from an external source (exogenous) acts locally upon the substance of the spinal cord and causes diffuse inflammatory degenerative changes therein. Such genuine myelitides are decidedly rare, and little definite is yet known as to their causes. We sometimes see the disease develop in previously healthy persons without, however, being able to demonstrate any toxic influence whatever. In other cases the blame is laid upon exposure to cold, repeated wettings, physical overexertion (such as military maneuvers), etc. In women the first symptoms of the disease sometimes follow childbed. These alleged causes, however, play at most the part of an exciting cause. It is wholly out of the question that sexual excesses, as was formerly believed, can of themselves lead to anatomical disease of the spinal cord; the same is true of severe emotional disturbances.

We may assume that the special cause of true acute myelitis is in all probability some infectious germ. This has been maintained for some time by me in regard to acute poliomyelitis (*vide infra*); but the evidence is increasing in favor of the infectious nature of acute transverse myelitis also. Thus I have seen a case follow a felon, and staphylococci were found in the cerebrospinal fluid, which was withdrawn by lumbar puncture. Fürstner found pneumococci in the foci of inflammation in a case of acute disseminated myelitis. In short, it is probable that we shall obtain still clearer ideas of the ætiology of acute myelitis by continued investigations. There is no doubt that infectious causes are to be considered in the rare cases of purulent inflammation of the cord (abscess of the cord); the same is true of the myelitides following acute infectious diseases (typhoid fever, smallpox, erysipelas, influenza, malaria, gonorrhea, etc.). But the question whether the disease in all these acute myelitides is due to the direct action of the bacteria which have penetrated the spinal cord, or is merely the result of the action of their toxins, is of fundamental importance. In my opinion both these possibilities must be considered. Personally, I am inclined to regard circumscribed, insular myelitis (transverse myelitis proper) as due directly to a bacterial invasion, and disseminated myelitis as due to the action of toxins. In this way, also, the transition of disseminated myelitis to "tract affections" is best explained.

That secondary myelitis may arise by direct extension of the inflammatory process from the vicinity has been definitely determined only in acute purulent meningitis (especially the epidemic cerebro-spinal form) and tubercular meningitis. In the other cases formerly classed under this heading, we have to do, as has repeatedly been said, mainly with purely mechanical consequences. The hypothesis that neuritic processes may extend to the cord (ascending neuritis) we have already (page 264) characterized as doubtful and practically unimportant.

Pathological Anatomy.—Macroscopical examination of the cord in its fresh condition shows no marked pathological changes except in a small number of cases. At the first glance, the spinal cord often seems almost completely normal, even if there have been severe spinal symptoms during life, and sometimes the opacities and adhesions of the pia, which often strike us at first, have no practical importance. If we test the consistency of the cord carefully by touching it, of course a change in it often strikes the practiced examiner, since the cord over a definite extent is either softer and more flexible, or, on the other hand, harder and firmer than normal. If we make a number of cross sections through the cord, we notice that the substance of the cord rises up more on section, that the outline of the gray matter is less distinct, and especially that the white matter is of a reddish-gray color, and that sometimes there is also a reddish, hyperæmic coloring of the gray matter. In some cases we can recognize small capillary hemorrhages with the naked eye. The more acute the disease the softer, as a rule, is the cord in the part diseased ("acute inflammatory softening"). If the disease is chronic, or if some time has elapsed since the expiration of the acute initial stage, the cord in the diseased spot is denser, firmer, sclerosed, and grayer in color on section, but it is not possible to determine precisely in the fresh cord the extent and degree of the disease.

The changes are much more plainly visible if we harden the cord in chromic acid, or Müller's fluid,[1] for at least eight or ten weeks. All the normal parts of the white matter of the cord assume a dark-green color, which is really due to the staining of the medullary sheaths by the chromic acid. The diseased portions, in which the medullary sheaths are mainly if not entirely absent, are thus often very sharply distinguished from the healthy, dark-green portions. Since similar, although less sharply defined, differences in color between healthy and diseased tissue are also noticed in the gray matter, the cross section of the cord, well hardened in chromic acid, usually gives quite a correct idea of the extent of the disease.

We obtain more precise disclosures, however, as to the form of the anatomical changes by microscopical examination. When made on the fresh, unhardened cord, it affords little information. The presence of numerous granular cells (*vide infra*) in fresh teased-out preparations is the only thing that is important, since they show with certainty the existence of a pathological change. If, however, we make thin cross sections of the hardened cord, and stain them with carmine or some similar staining fluid, even the naked eye notices at once a marked difference between the diseased and the healthy tissue, since the former, which is always richer in neuroglia, has a much darker staining, and thus is distinguished from the brighter normal tissue. The microscopic examination now shows that in the diseased parts the normal nerve tissue has been almost wholly or at least partly destroyed. Only occasionally do we see nerve fibers of normal appearance remaining here and there. In other places the fibers that are still visible are smaller and atrophied, and the axis cylinders have in part lost their medullary sheaths or are swollen. The changes in the ganglion cells are harder to follow, but in more advanced cases they also show marked signs of destruction; they are contracted, rounder, and have lost their processes. The increase of the neuroglia tissue corresponds to the destruction of the nerve substance. The meshes of the neuroglia extend and swell, so that the space formed by the destruction of the nerve tissue is in great measure taken up by neuroglia. The older the process, the firmer and more fibrous is the neuroglia. The nuclei of the neuroglia increase in number, and we often find a very great increase in those peculiar, flat cells with many processes, first described by Deiters and named after him—the so-called " Deiters' spider cells." The fatty granular cells are also easily recognized in hardened preparations, provided they are not treated with alcohol. They lie in the interstices between the meshes of the neuroglia, and are especially numerous about the vessels. They are to be regarded either as white blood corpuscles or perhaps as endothelial cells from the sheaths of the vessels, which have taken up the fat from the disintegrated nerve substance. If, therefore, the process be still fresh, or if it be still advancing, the fatty granular globules are to be met with in great numbers, while in old, sclerosed nodules only a few of them, or scarcely any, are to be found. The changes in the vessels are usually very striking. They are often dilated and congested. Here and there there may be hemorrhages. The vascular walls are thickened, especially in old cases, and sometimes have become peculiarly homogeneous—" hyaline degeneration"; and a large accumulation of nuclei, lines of fatty granular cells or peculiar epithelioid cells, may be found about the vessels. The so-called corpora amylacea are sometimes present in chronic cases in great numbers, and sometimes they are only scanty. Their significance and their genesis are still unknown.

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[1] [The formula for Müller's fluid is as follows: Two and a half parts of potassium bichromate, one part of sodium sulphate, and one hundred parts of water.—K.]

The extent of the whole process varies very much in different cases. It is important to distinguish true transverse myelitis from the disseminated form (myelitis disseminata). In transverse myelitis we find one main focus of myelitis, which extends in a diffuse manner over the greatest part of the transverse section of the spinal cord, and which may reach upward and downward for a space of 5 to 10 cm. or more. The dorsal portion of the cord is most frequently affected (dorsal myelitis), the upper half being usually most involved, but in some cases the lower half is chiefly affected. Nearly the whole of the dorsal cord is often the seat of a diffuse inflammatory affection, which, of course, differs in extent at different levels. In other much rarer cases the chief focus of disease is in the cervical cord (cervical myelitis), somewhat more frequently in the lumbar cord (lumbar myelitis). We often find small, distinct foci in the vicinity of the main focus. In all severe and long-continued cases there develops later a systemic ascending and descending secondary degeneration (*vide infra*).

Acute disseminated myelitis has a different form of anatomical distribution. It occurs in many small foci which may extend almost throughout the whole length of the cord. Individual columns of the cord, especially the posterior columns, are often chiefly affected. Occasionally foci of inflammation are found to exist at the same time in the medulla, the pons, and the brain. The frequent occurrence of optic neuritis (*vide infra*) is of great diagnostic importance. Acute disseminated myelitis appears apparently spontaneously, or secondary to a preceding infectious disease. In all probability toxic substances (toxins) play the most important part in the production of this disease. The more chronic this action is, the more the disease acquires the character of a simple tract degeneration.

The Individual Symptoms of Myelitis.—The course of transverse myelitis differs so much in different cases that it is impossible to give a picture of the disease which will be generally applicable. According as one or another part of the cord is involved, the clinical symptoms will exhibit many differences. The following description will, therefore, be devoted first to the single symptoms, and will give the inferences which according to the present state of our knowledge, may be drawn from their presence as to the seat and the extent of the anatomical process; not until then will we endeavor to distinguish the different clinical forms of myelitis.

1. SYMPTOMS OF MOTOR PARALYSIS.—Symptoms of motor paralysis are not only the chief symptoms, as a rule, in well-developed myelitis, but are often the first sign of the beginning of the disease. A weakness of the legs, frequently progressing to a complete paralysis, develops very rapidly in acute myelitis, and more slowly in those varieties which take a more chronic course.

Since the paths for the conduction of voluntary motion are situated, as we have seen, in the antero-lateral columns of the spinal cord, we conclude, in every spinal disease where symptoms of paralysis are present, that there is an implication of the lateral columns. Since, furthermore, in transverse myelitis the whole cross section of the cord is more or less involved, the paralysis also extends to the two halves of the body; paraplegia is the characteristic form of paralysis in transverse myelitis. Paraplegia of the lower extremities may, of course, arise wherever the myelitis is situated, whether in the lumbar, dorsal, or cervical region; but the upper extremities necessarily remain entirely free in every dorsal or lumbar myelitis. The occurrence of paretic symptoms in the arms and the final development of a brachial paraplegia, point with certainty to an implication of the cervical region, to a cervical myelitis. If the symptoms of paralysis are not alike in the two corresponding extremities, but are more marked on one side than on the other, the anatomical affection must also be more intense on that side of the cord than on the opposite side. If the motor pyramidal tract proper is alone affected, then the paralysis of the legs is not complete, but affects principally flexion of the leg and movements of the foot. If a complete paraplegia of the lower extremities exist, we can always infer that there is an extensive involvement of the antero-lateral tracts.

2. SYMPTOMS OF MOTOR IRRITATION.—Symptoms of motor irritation of various sorts are often seen, both at the beginning and during the whole

course of myelitis. Single twitchings come on spontaneously in the limbs, which are at the same time paralyzed, or at least paretic, and these twitchings are short and rapid or slow and persistent. The thighs are drawn up on the abdomen, or there are severe spasms of the extensors. The interpretation of these symptoms is not always easy. It is often particularly hard to decide whether they are the result of a direct irritation of motor fibers in the cord, or whether they represent reflexes (*vide infra*). The value of the symptoms of motor irritation for the localization of the disease is accordingly slight, but, of course, in these cases we must consider chiefly the motor tracts in the lateral columns.

3. Ataxia.—There can be no doubt that actual ataxia also occurs in transverse myelitis, but it is rarely pronounced because the coexisting spastic or paralytic symptoms, which are almost always present, of course conceal the ataxia. We therefore see evident ataxic disturbances rather in slow cases which do not speedily become paraplegic, or sometimes not until the disease improves. In regard to the localization of the disease, we may always conclude, when there is ataxia, that the posterior columns are involved. Very marked ataxia of the arms and legs occurs therefore in acute disseminated myelitis of the posterior columns.

4. Disturbances of Sensibility.—The disturbances of sensibility usually appear to a marked degree only in the later stages of the disease. At the outset we generally notice merely mild symptoms of sensory irritation in the legs, such as formication, prickling, numbness, a woolly feeling, etc., while severe pain is hardly ever present in transverse myelitis, and hence it always points rather to some affection of the vertebræ or the meninges. In very acute myelitis there may be at first severe local pain in the back. Slight diminution of sensibility is often to be made out early on careful examination, but, in many cases, the sensibility remains for a long time wholly, or almost wholly, intact, either because the localization of the disease spares the sensory portions of the cord, or because the sensory paths of conduction are more resistant, or can act vicariously for one another to a higher degree. The more carefully we examine, the earlier shall we be able, as a rule, to find at least partial disturbances of sensibility. From the results of these examinations, we can, according to what has been previously said (page 156), draw some conclusions concerning the localization of the myelitic disease. If there is mainly a disturbance of the sense of touch, muscular sense, and the sense of pressure, we may conclude that the posterior columns are chiefly affected, as I have observed particularly in cases of disseminated myelitis. Disturbances of the sensibility to pain and temperature indicate an involvement of the gray posterior horns. To what extent involvement of the lateral tracts can produce disturbances of sensibility (analgesia and thermoanæsthesia?) has so far been insufficiently investigated. In severe transverse myelitis we always find marked disturbances of sensation, occasionally even complete anæsthesia. In some cases a retardation of pain perception, or a marked hyperæsthesia to painful stimuli (pin pricks), is observed.

The disturbance of sensibility gives important service in estimating the height at which the affection in the cord is situated. If we search on the trunk for the line where the cutaneous sensibility becomes normal, we may from this determine the upper boundary of the myelitis in accordance with the data furnished on page 289, but since each cutaneous region, as has been said, is supplied with sensory fibers from at least two or three spinal segments, we must look for the upper boundary of the disease not simply in the segment corresponding to the upper boundary of the anæsthetic area, but one or two segments higher. In myelitis in the lumbar region the disturbance of sensibility reaches to the umbilicus, or even a little higher; in myelitis in the lower dorsal region it reaches about to the lower end of the sternum; in myelitis in the upper dorsal region to the level of the axillæ; and in cervical myelitis the sensibility of the upper extremities is also impaired, but complete anæsthesia is very rare.

5. Cutaneous Reflexes.—As is generally assumed, the reflex arcs in the cord are found at about the same level as the centripetal sensory and the centrifugal motor fibers. They are also connected with fibers which come from above and have properties of reflex inhibition. If these fibers above the reflex arc are irritated, the reflex is thereby impaired; but if the conduction be broken in these fibers, the reflex activity appears increased, the reflex comes on with a weaker irritation, and the contraction is more vigorous. If the reflex arc itself be broken at any ponit, the reflex must disappear.

The data from the examination of the patient may generally be harmonized with this scheme, although, of course, the reality probably shows more complicated conditions. In extensive lumbar myelitis, by which the reflex path in the lumbar cord is broken, the cutaneous reflexes in the lower extremities must be diminished or absent. In these cases the loss of sensibility runs about parallel to the diminution of the reflexes. In dorsal and cervical myelitis, however, the reflex arc in the lumbar cord remains unimpaired, but the conduction of sensory impressions to the brain may very well be interrupted. In these cases the cutaneous reflexes are retained, even when there is anæsthesia; or, if the reflex inhibitory influences be removed, they are decidedly increased. [The Babinski and Oppenheim reflexes are often present.—K.] The cutaneous reflexes in the legs, however, may also be diminished or delayed in myelitis of the lower dorsal cord, in which case we must imagine, as a rule, a secondary loss of excitability of the fibers and cells presiding over the reflex. Concerning the details of reflex contractions, compare what has been previously said on page 207. It is also always of interest to examine the cremaster reflex, the abdominal reflex, etc. If the reflex paths described above (page 293) be interrupted, the reflexes, of course, must be absent. However, these reflexes often disappear even when the disease is localized higher up, just as they do in cerebral hemiplegia (*q. v.*), as we shall learn later on.

6. Tendon Reflexes.—The same rules generally hold in judging of the tendon reflexes as are to be considered in judging of the condition of the cutaneous reflexes. We know that the reflex arc of the patellar reflex is at the level of the second to the fourth lumbar segments (page 293). If the entrance zones of the posterior sensory roots or the ganglion cells in the anterior horns be diseased at this level, the patellar reflex must disappear. The Achilles tendon reflex and the ankle clonus have their reflex arcs in the sacral segments of the cord. The Achilles-tendon reflex is always absent in extensive disease of the posterior columns and of the gray matter in the corresponding portion of the lumbar cord. The absence of the tendon reflexes in the

lower extremities is therefore one of the most important points, besides the other symptoms, for the diagnosis of myelitis of the lumbar cord. In almost all inflammations above the lumbar cord—that is, in dorsal and cervical myelitis—there is, however, a very decided increase of the tendon reflexes, the result, as we must suppose, of the loss of the reflex inhibitory influences. We have much reason to assume that the fibers which influence the condition of the tendon reflexes run chiefly in the pyramidal tract of the spinal cord. We may therefore assert that, with a considerable increase of the tendon reflexes in the lower extremities, the seat of the myelitis must be above the lumbar cord—that is, in the cervical or dorsal cord—and that in these cases we have to suppose that the lateral columns, particularly the region of the pyramidal tracts, are chiefly implicated. In cervical myelitis the tendon reflexes in the upper extremities are often considerably increased.

We have already said, on page 209 *et seq.*, what is necessary in regard to the different signs of the increased tendon reflexes, the exaggerated patellar reflex, ankle clonus, the periosteal reflexes, etc. The peculiar character which the paralysis of the legs assumes from the fact that the lateral pyramidal tracts are principally involved (spastic paraplegia) will be described more fully in the chapter on "spastic spinal paralysis" (*vide infra*).

7. Disturbances in the Bladder and Rectum.—Disturbances in micturition are one of the commonest symptoms of myelitis. According to the researches of L. R. Müller, which were conducted at our clinic, the emptying of the bladder is a reflex action conveyed only by the sympathetic nervous system, independently of the spinal cord. In the healthy adult, however, the bladder reflex is called forth "voluntarily" by the cerebrum. The fibers which convey the reflex stimuli pass downward throughout the entire length of the spinal cord, and leave the latter only at the conus medullaris. Therefore in all transverse affections of the spinal cord dysuria (difficulty in urination) or complete retention of urine appears first. If the bladder be filled to the maximum, the overflow urine generally dribbles away (ischuria paradoxa). After some time, however, the reflex mechanism for emptying the bladder begins to function of its own accord. The bladder then empties itself spontaneously in shorter or longer intervals (about every half hour to two hours). As a rule, the patients are aware of the coming on of the reflex, since the sensory stimuli conveyed to the bladder by the sympathetic nerves are still transmitted to the brain by the spinal cord. In isolated cases this path for the conduction of sensory impulses is also interrupted, and the bladder is then emptied wholly unconsciously. The inhibition of this reflex is possible only by contraction of the (striated) compressor urethræ muscle, which is also generally paralyzed in cases of myelitis. In patients suffering from spinal-cord disease, we can very frequently produce contraction of the bladder with involuntary micturition by stimulating the skin of the thighs, perineum, etc.

The clinical significance of disturbances of the bladder in myelitis, and in many other diseases of the cord, apart from the great distress and discomfort for the patient, lies in the fact that they very often—almost always in severe cases—give rise to the development of cystitis. In retention of urine the use of the catheter, by which inflammatory irritants are often brought into the bladder, in spite of all attempts at disinfection, leads to decomposition of the urine and to cystitis. In addition, the presence of stagnating and decomposing urine in the urethra may sometimes be the cause of the entrance of these irritants into the bladder. If cystitis has developed, it may be followed under some circumstances by pyelitis and purulent pyelonephritis (*q. v.*), which conditions are often the immediate cause of death from the sequelæ connected with them, such as fever—sometimes associated with chills—general weakness, and emaciation.

Defecation is also disturbed in many cases of myelitis. There is usually constipation at first, since the reflex mechanism for emptying the rectum is disturbed in a similar way to that of the bladder. Paresis of the abdominal muscles is an important factor here. Sometimes the constipation reaches such a degree that the bowels move only at intervals of one or two weeks. Defecation occurs later on in a purely reflex manner (so-called incontinentia alvi). It cannot be voluntarily inhibited if the external sphincter of the anus be paralyzed, since the spinal center of the latter is located in the lowest sacral segment of the spinal cord.

In conclusion we may mention, as an addendum, that the sexual functions are often considerably disturbed in many cases of myelitis, and finally they may be wholly lost.

8. TROPHIC DISTURBANCES.—The trophic condition of the paralyzed muscles affords extremely important points for diagnosis. In cervical and dorsal myelitis the motor centers in the lumbar cord for the muscles of the legs remain intact; the paralyzed muscles, therefore, do not exhibit rapid atrophy, and, above all, retain their normal electrical excitability. A certain degree of atrophy of the muscles is, nevertheless, frequently found in such cases; this is due in part to diminished nutrition, in part to a lack of movement ("atrophy of disuse"). In flaccid palsies, where reflex stimulation is also lacking, or very much diminished, atrophy of the muscles is always more pronounced. But if we find in myelitis a marked atrophy, with reaction of degeneration in the muscles of the lower extremities, we can from this draw a definite conclusion that the anterior gray cornua or the fibers of the anterior roots in the lumbar cord are affected (see page 222). In an analogous fashion degenerative atrophy with reaction of degeneration in the muscles of the upper extremities points to an affection of the anterior gray matter in the cervical cord.

Trophic disturbances in the skin are also frequent. We often find the skin dry, hard, with a scaly epidermis, and the nails thickened and brittle. Exceptionally there are eruptions of herpes, urticaria, etc. Vasomotor disturbances also occur. Sometimes the paralyzed extremities show a mottled, cyanotic reddening, and feel cold. Slight œdema is quite frequently present in the paralyzed parts. Disturbances of the sweat secretion are not infrequent. We find either that it is absent or that there is a great increase in it, so that the paralyzed parts are constantly moist. All these symptoms have no value at present for the special topical diagnosis.

The frequent occurrence of bedsores in the sacral region, over the glutæi, or more rarely over the trochanters, on the feet (heels), or the inner side of the knees, is of great practical importance. Although trophic and vasomotor influences may play a part in their origin, still, their ultimate cause is always to be found in external conditions, pressure, uncleanliness, etc. The more faulty the care of the patient is, the easier bedsores arise. With completely paralyzed and anæsthetic patients, with incontinence of urine and feces, of course they sometimes cannot be wholly and permanently avoided, even with the most careful management. The extent which a bedsore may reach is sometimes absolutely frightful. A large part of the sacrum may be laid bare, after the overlying soft parts and the periosteum have become gangrenous and been thrown off. In like manner deep bedsores extending to the bone are occasionally found on the heel. Such a bedsore is sometimes the starting point of a progressing phlegmon. If we fail to check the spread of the phlegmon, a general septic condition develops (fever, chills, diarrhea, splenic enlargement, etc.), from which the patient finally succumbs.

9. DISTURBANCES IN THE REGION OF THE CEREBRAL NERVES are entirely absent in most cases of transverse myelitis. In rare cases of cervical myelitis the process may gradually extend upward and give rise to bulbar symptoms. We sometimes see changes in the pupils in cervical myelitis, such as inequality and spinal myosis. Lastly, the combination of acute myelitis with optic neuritis (*vide infra*) is of great interest.

Different Forms of Myelitis. Acute and Chronic Myelitis. Myelitis acuta disseminata.—The whole picture of transverse myelitis in its different forms may be constructed from the symptoms described in detail in the preceding paragraphs. We can usually determine without difficulty, at least approximately, the seat and extent of the disease. If we group the chief symptoms of the different forms of myelitis together, according to the location of the disease in the cord, they are as follows:

Cervical Myelitis.—Paraplegia of the legs, combined with more or less extensive disturbances in the upper extremities, and eventually disturbances of sensibility over a like extent. At times atrophy of single muscular regions in the arms. No marked atrophy in the legs. Increased tendon reflexes and spastic symptoms in the legs and often in the arms. Cutaneous reflexes in the legs retained, and sometimes even increased. Disturbances of the bladder and rectum. Sometimes changes in the pupils.

Dorsal Myelitis.—Upper extremities free. Motor, and eventually sensory, paraplegia of the legs, without pronounced muscular atrophy; preservation of the electrical excitability of the nerves and muscles. Increased tendon reflexes and pronounced spastic symptoms in the legs (especially in myelitis in the upper dorsal cord); cutaneous reflexes retained, sometimes increased. Disturbances of the bladder and rectum.

Lumbar Myelitis.—Upper extremities free. Motor, and eventually sensory, paraplegia of the legs. Cutaneous and tendon reflexes in the legs diminished or absent. Generally rapid degenerative muscular atrophy in the legs with electrical reaction of degeneration. Disturbances of the bladder and rectum. In myelitis of the *lower sacral cord* the paralysis is confined chiefly to the distribution of the sciatics and the bladder and rectum. The disturbance of sensation is found on the posterior aspect of the thighs, the buttocks, and the genitals (page 290).

The above division of the different forms of myelitis refers only to the different locations of the disease and the difference in symptoms thereby produced, so that we must further discuss the division of myelitis into an *acute* and a *chronic* form. This division is made solely by observing the course of the disease, or from the statements in the history of the case as to duration; but we must bear in mind that this refers only to the first stage of the disease, to the rapidity with which the myelitic symptoms develop at the start. In most cases of genuine primary myelitis the myelitic symptoms come on very suddenly, lead in a very short time (sometimes in a few days or a few weeks) to the most complete paralysis (paraplegia, vesical paralysis, anæsthesia, bedsores, etc.), and advance irresistibly, causing death in a short time (from four to twelve weeks). Such cases are, of course, called acute myelitis. We sometimes see, however, the disease beginning acutely, and rapidly developing a more or less complete paraplegia, which after some time remains entirely stationary, or at least shows only extremely slight further changes. Such cases must also be classed definitely as acute myelitis, although they present later a chronic type of spinal disease, which sometimes lasts for many years; but they are also undoubtedly cases of acute myelitis, ending in relative recovery—that is, in recovery with a scar or defect in the cord. We can call only those cases actual chronic myelitis which are chronic from the first—that is, which begin slowly and progress gradually—but such forms of actual chronic myelitis are very rare, for the not very rare cases which seem to belong under this heading during life usually prove at the autopsy, at least as far as our experience goes, to be something different—multiple sclerosis, tumor, combined columnar or systemic disease, etc.

A true circumscribed chronic transverse myelitis, so far as we know, never occurs. Certain chronic tract degenerations caused by exogenous (toxic) substances could more properly be classed with the chronic myelitides. The spinal degeneration develops more or less acutely, depending upon the mode of action of these toxins. Occasionally, also, an acute initial onset may be followed by a slowly progressing course. At any rate, in our definition of acute and chronic myelitis, we must pay especial attention to the clinical course and an accurate history, for the pathological lesions alone cannot be conclusive. The pathologist cannot determine whether a given transverse sclerosis of the cord is the result of a process chronic from the first or of a long-past acute process.

It is important to distinguish two forms of acute myelitis—the true transverse myelitis (circumscribed, focal myelitis) and disseminated myelitis. This distinction, at the present state of our knowledge, is not very easy, since disseminated myelitis, as a rule, runs its course under the clinical picture of a transverse paraplegia. There are, however, cases (especially those seen after acute infectious diseases) in which the clinical picture resembles more closely a simple or combined tract degeneration. Acute ataxia with loss of tendon reflexes and marked disturbances of the bladder, in other cases combinations of ataxia with paresis and spastic conditions, have been most frequently observed. Of especial diagnostic importance is the occurrence of optic neuritis, which not infrequently, in conjunction with headache, vertigo, vomiting, and visual disturbances, ushers in the entire clinical picture. The paraplegic symptoms develop a short time after. In spite of the fact that the sight is practically restored, ophthalmoscopic examination always shows distinct changes in the optic nerve. This examination should therefore not be neglected in any case of acute myelitis. Recovery from acute myelitis is perhaps not impossible, but it is rare. Some of the "cured cases of myelitis," formerly reported, were probably unrecognized multiple neuritis, compression of the spinal cord, etc. Some recent experiences have taught me, however, that there is a curable form of acute disseminated tract myelitis, but these cases are very rare.

We may speak somewhat more frequently of relative recovery if the severe initial symptoms subside in part and change to a mild, stationary, morbid state. The fatal termination of acute or chronic myelitis is the result of the general weakness which finally sets in; or it comes from cystitis or pyelonephritis, both of which are often combined with pyæmic conditions; or from extensive bedsores, with secondary sepsis, or, finally, from some complication, such as tuberculosis or acute diseases.

Diagnosis.—The diagnosis of myelitis is always made by considering the whole course of the disease (accurate history!) and the existing group of symptoms. The possibility of a compression of the cord, by disease of the vertebræ, etc., must be excluded by a careful examination of the vertebral column (Röntgen-ray examination, if possible), and by consideration of the course of the disease. We must also be sure that the existing symptoms do not correspond to a definite typical disease, or a systemic disease, but that they can agree only with the assumption of a diffuse transverse disease at a certain point in the cord, to be made out accurately according to the symptoms. An accurate history, particularly of the onset of the disease, is of the greatest importance in determining the variety of the affection. If trauma, syphilis, etc., can be excluded, we inquire in the first place for other toxic infectious causes (preceding infectious diseases, tonsillitis, intestinal affections, etc.) and for possible rheumatic causes (catching cold, etc.). In all apparently spontaneous spinal paralyses appearing in an acute manner (that is, in a few days, or, at most, in a few weeks), the assumption of an acute inflammatory process of the spinal cord is almost the only possible one. An accurate examination will then generally show whether we are dealing with a so-called acute poliomyelitis (*q. v.*) or a disseminated acute myelitis extending either transversely or along the spinal tracts. Particular attention should be paid in distinguishing this disease from acute polyneuritis (*vide supra*), since it is of the greatest prognostic importance. Attention should be directed, above all, to the early occurrence of pain and tenderness in the affected extremities.

The diagnosis is much more difficult in the slowly developing paraplegias (that is, those developing within several months or even years). If spinal disease can be excluded, and a chronic affection of the cord itself can, therefore, be assumed, the diagnosis of a "chronic myelitis" is the most probable; but, as we have already emphasized above, the occurrence of circumscribed chronic myelitis has not as yet been positively demonstrated. Cases such as are cited above at autopsy generally turn out to be either true multiple sclerosis, tumors (glioma, etc.), or so-called combined tract disease. In the description of all these diseases we will once more return to the likeness sometimes observed between them and transverse chronic myelitis.

Treatment.—Although therapeutic endeavors are rarely permanently and completely successful, still, we can often relieve the suffering and delay the end. In recent cases absolute rest and quiet of the patient are the first requisites.

Careful diaphoresis may be attempted; internally we administer the salicylates, iodid of potassium, etc. If syphilis cannot be definitely excluded in the ætiology, inunctions are prescribed daily (gr. xlv to lxxv = gm. 3 to 5 of mercurial ointment) and gr. xxijss. to xxx = gm. 1.5 to 2 of iodid of potassium are administered internally daily. [These doses of iodid are often insufficient (page 421).—K.] We sometimes see decided improvement from this; but in some cases, of course, the result is uncertain, or the treatment seems to exert even an unfavorable influence upon the disease. In the latter case we must stop it at once.

In the later stages of the disease the chief methods of treatment are electricity, baths, and other hydrotherapeutic measures. We alternate these. New attempts at cure raise the patient's courage and hope afresh.

Electricity may give symptomatic improvement in many cases, but it causes recovery only exceptionally. In severe and hopeless cases, however, it is at least the best means of consoling the patient. The constant current has the greatest therapeutic value. We use large electrodes placed on the vertebral column, and pass not too strong a stabile or slowly labile current through the cord for about four or five minutes, chiefly through the region where we suppose the seat of the disease to be. We usually take the ascending current, and alternate with the two poles on the diseased part. We should avoid reversals and great variations in the strength of the current. We associate with this peripheral galvanization and faradization of the muscles and nerves of the paralyzed extremities. Single symptoms sometimes deserve special attention —faradization of the skin in anæsthesia, galvanization of the bladder in vesical weakness, etc. The electrical treatments should take place daily or every other day. If we would be successful, the treatment must be kept up persistently for months.

The treatment of myelitis by baths, if prudently used, may also be of evident service. Even simple tub baths, such as can be had in almost every household, do good service under some circumstances. The chief rules are never to make the baths too warm—about 88° or 95° F. at most (25° to 28° R.)—to limit them at first to ten or fifteen minutes, and to give them at first not oftener than three or four times a week. Sometimes hot baths (95° to 100° F. [28° to 30° R.] or more) seem to act well, but in other cases they certainly are injurious. If the baths are well borne, they may be employed daily. We should be most cautious in incipient and still advancing cases. The best action of the simple warm bath is seen in chronic myelitis with predominant spastic symptoms. In these cases the duration of the baths may be increased to an hour or more. Sometimes the baths work still better when certain substances are added, especially if we use salt baths, which are made by the addition of 5 or 10 pounds of common salt (Stassfurt salt), or 4 to 6 pounds of brine salt, or 1 to 3 quarts of brine to the water. The neurogen baths and the artificial carbonic-acid baths, which have been recently introduced by several manufacturers, and which can be readily installed in every house, are very much to be recommended.

If we can send patients in easy circumstances to a health resort, the carbonic-acid thermal salt springs at Oeynhausen and Nauheim are most suitable for this purpose; also the baths at Kissingen, Wiesbaden, and Baden-Baden, the thermal baths of Ragatz, Teplitz, Wildbad, Gastein, etc.

A methodical hydrotherapeutic treatment sometimes gives quite good results; but in these cases we should wholly avoid all the more heroic treatments, such as douches, violent rubbing, and very cold baths, and employ only short, cool, full or half baths or packs, or mild cold sponging. Hydrotherapeutics are usually combined with electricity.

We can expect but little success from internal treatment, but it cannot be dispensed with in practice. A trial of strychnin (subcutaneously or by the mouth) is most to be recommended, and also ergotin, nitrate of silver, and especially iodid of potassium.

The general hygienic and symptomatic treatment is very important. The diet should be strengthening but easily digestible. Large amounts of spirits, much smoking, much tea and coffee, etc., are to be avoided. If the patient becomes bedridden, we must employ the utmost care to get a good bed in order to guard against bedsores. In severe cases, especially where there are disturbances of sensibility, a water cushion is most desirable. The patient's position must also be frequently changed, and the sacral region must often be washed and rubbed. Every incipient bedsore must be very carefully treated, in order to prevent its spreading, by Peruvian balsam ointment (1 to 30), dermatol, salicylate plaster, or mercurial plaster, which acts very well. When the bedsore is very extensive the continuous bath is frequently the only remedy by which a cure is still possible.

If there is retention of urine and the patient has to be catheterized, the most extreme care must be employed in cleaning and disinfecting the catheter, or else cystitis will develop in a few days. If a cystitis already exists, then urotropin, salol, and potassium chlorate are the most effective internal remedies. It is best in severe cases to wash out the bladder regularly with boracic-acid solution, permanganate of potassium, and like remedies. In milder cases we may try chlorate of potassium internally. With incontinence, men are supplied with a suitable urinal. Women with incontinence must be kept very clean (frequently changed draw sheets, absorbent pads to absorb the urine, etc.).

Constipation must be met according to the general rules. We should be sparing with cathartics, and try to make an appropriate diet and enemata suffice. If there is severe pain, subcutaneous injections of morphin are unavoidable, but we always delay this as long as possible, although finally we let the dose of morphin be unlimited in hopeless cases.

CHAPTER VI

MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD

(Disseminated Nodular Sclerosis. Sclérose en plaques)

Ætiology and Pathology.—Multiple sclerosis of the central nervous system is a special chronic form of disease, whose anatomical basis consists in the development of numerous disseminated "sclerotic nodules" (*vide infra*) in the brain and cord. We know practically nothing as to its ætiology, for the significance of exposure to cold, overexertion, and mental emotions, sometimes given as causes of the disease, is wholly doubtful. It is also very improbable that syphilis plays any part in the ætiology of multiple sclerosis. P. Marie claims to have seen the disease come on very frequently as a consequence of some acute infectious disease (typhoid fever, scarlet fever, etc.), and therefore he suspects that the different foci of disease have an infectious inflammatory origin; but our own experience does not permit us to confirm this. It is true that after an acute infectious disease an acute disseminated myelitis may occur, which, for very evident reasons, may resemble multiple sclerosis very closely clinically, but it is an essentially different morbid process from the latter. Nor, similarly, does our own experience pronounce in favor of a relation between multiple sclerosis and chronic intoxication, as Oppenheim supposes; on the contrary, in the majority of our own observations we could find absolutely no material external cause, in spite of careful inquiry. We are therefore disposed to regard the disease as of endogenous nature, as a multiple gliosis due to congenital abnormal predisposition. The anatomical data also harmonize very well with this view. Any injury preceding the development of the disease, acute diseases, intoxications, emotional disturbances, etc., would therefore act at most as exciting causes. Particular attention is called to trauma as a cause. I have already seen a series of cases in which the initial symptoms occurred after a severe fall on the back, etc. This fact is of importance in accident practice.

An hereditary predisposition seems to be present in only a few cases. The affection occurs chiefly in youth, somewhere between the ages of eighteen and thirty-five, but we have ourselves seen one case, which came to an autopsy, in a man of sixty. The more carefully we inquire, the more frequently will we learn that the initial symptoms of the disease were noticed at puberty, or even before that time. Isolated cases of multiple sclerosis have been observed in children, but it is certainly a rare occurrence to find this disease during childhood. No material distinction has been made out as to sex.

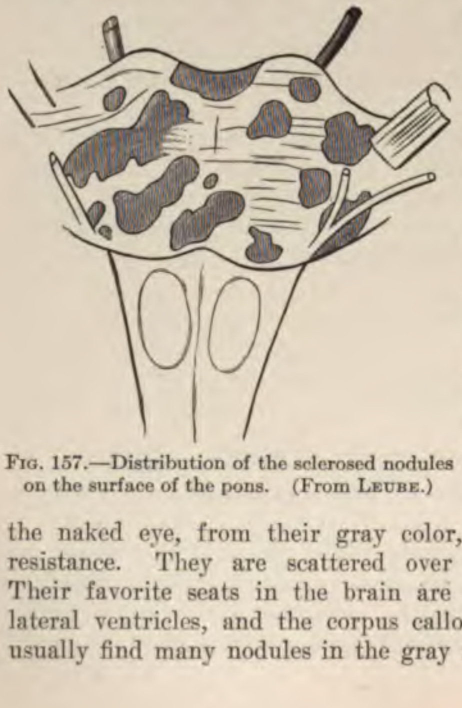
FIG. 157.—Distribution of the sclerosed nodules on the surface of the pons. (From LEUBE.)

In regard to the development of the different sclerotic nodules, nothing definite has been established at present as to their genesis. The suspicion has repeatedly been uttered that the disease is due to primary changes in the vessels, but no proof of this can be given. The nodules are in part easy to recognize with the naked eye, from their gray color, and we can also feel an increased resistance. They are scattered over the whole central nervous system. Their favorite seats in the brain are the centrum ovale, the walls of the lateral ventricles, and the corpus callosum, but on careful examination we usually find many nodules in the gray matter of the central ganglia and the cortex; the nodules are also quite abundant, as a rule, in the pons, less frequent in the medulla, and very abundant and variously distributed in the cord (see Figs. 157 and 158), chiefly in its white substance. Examined microscopically, the nodules consist of an abundant, reticulated, fibrillary neuroglia tissue, in which the nerve fibers are lessened in number, but, as a rule, not wholly destroyed; on the contrary, we usually find many axis cylinders preserved in the nodules, as Charcot first found and as has often been confirmed since by other observers. This fact is important clinically, because it explains why multiple sclerosis only rarely, or only in its advanced stages, leads to complete paralysis. It also indicates, as it seems to us, that the process does not start from the nervous tissue, but from the interstitial neuroglia tissue. It also explains the remarkable fact that the ordinary secondary degenerations are almost always absent, in spite of the presence of many nodules in the spinal cord. In the vessels we usually notice thickening of the walls, which may be surrounded by neuroglia cells. I regard these changes as of secondary nature. Fatty granular cells are often to be found in fresh cases. It is also remarkable that the foci of multiple sclerosis rarely show any tendency to contract. In this point there is a marked difference between the inflammatory and neoplastic gliomatous foci. The general contour of the spinal cord is therefore but little altered, in spite of the presence of numerous sclerotic foci. Very characteristic also is the frequently sharp linear demarcation of the sclerotic foci from the normal tissue. This also is in favor of a degenerative metaplasia of the tissues rather than a true inflammation. There is, in our opinion, a sort of "gliomatous degeneration" of the affected areas of the tissue. For this reason, also, there are certain relations between multiple sclerosis and central gliosis (*q. v.*).

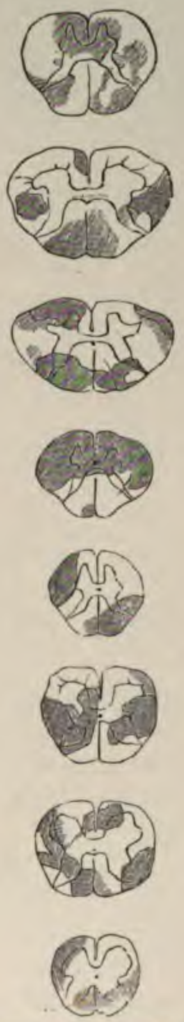
FIG. 158.—Example of disease of the cord in multiple sclerosis. The dark portions are the parts diseased.

Clinical History.—From the variations which the number and the localization of the nodules show, it may be understood from the outset that a picture of the disease which represents all cases cannot be drawn; but many cases show so characteristic a group of symptoms that the diagnosis can often be made with considerable certainty. We are indebted, above all, to Charcot for our knowledge of this typical clinical picture, although, for very evident reasons, he was at first able to diagnosticate only the fully developed cases with very pronounced symptoms. At present, we know that the classical clinical picture described by Charcot is found in its entirety only in a comparatively small number of cases. The disease occurs much more frequently in a less complicated form, which, with our present knowledge, can nevertheless generally be easily recognized and diagnosticated. However, there are many in the lateral tracts. The occasion of the oscillatory tremor is still wholly unknown. In my opinion, it is due to a disturbance of the mutual reflex relations of the antagonists to each other.

There are still, however, some other symptoms in addition to those already described which must be enumerated. They are not prominent symptoms in the general clinical picture, but are nevertheless of the greatest diagnostic importance. The cutaneous reflexes deserve the first consideration. Whereas, the tendon reflexes are almost invariably increased, we often find a marked diminution of the cutaneous reflexes. The plantar reflex is often very slight, but much more remarkable is the loss of certain other reflexes—namely, the cremaster reflex and, above all, the abdominal reflex. Since my attention has been called to this fact, I have found it confirmed almost without exception in more than one hundred cases. The loss of the abdominal reflex occurs very early. It is of great importance, as this disease generally occurs in young people with tense abdominal muscles, where the reflexes are normally hardly ever entirely absent. A second extremely important symptom is the pallor of the temporal half of the optic disc, which can be frequently demonstrated by ophthalmoscopic examination (Uhthoff and others). This objective symptom often occurs very early, though the patients themselves are not aware of any marked visual disturbance. Complete atrophy and blindness occur finally in a comparatively few rare cases. On the other hand, however, the contrast between the marked ophthalmoscopic changes and the comparatively insignificant disturbance of vision which is occasionally found is remarkable. This condition is probably due to the conservation of the axis cylinders. Nevertheless, on careful examination, we find not infrequently a contraction and limitation of the visual field, disturbances of color vision, etc. It is very curious that occasionally, at the onset of the disease (*vide infra*), total blindness of one eye occurs for a short time, and then disappears again. In rare cases optic neuritis and choked disc have been observed, which are due perhaps to retrobulbar sclerotic foci. Disturbances of the ocular muscles associated with double vision not infrequently occur, occasionally even as the initial symptom. The ocular palsies are either unilateral or bilateral. Occasionally we find paralysis of associated movements (foci in the pons). Loss of pupillary reflex

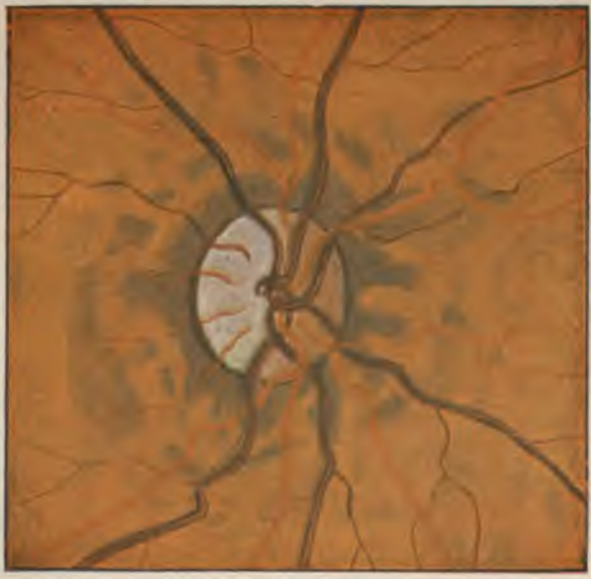

FIG. 159.—Pallor of the temporal half of the optic nerve (beginning optic atrophy in multiple sclerosis).

spastic-ataxic gait, entirely distinct from the pure ataxic gait found in tabes.

The ataxia of the gait is due (unlike the ordinary tabetic gait) not to the uncertainty of the legs, but to the swaying of the whole body, as in cerebellar ataxia. If the spastic symptoms become more prominent, the gait also becomes more spastic, but the individual steps are always quick and long on account of the absence of paresis. In many cases with slight ataxia and marked hypertonicity of the muscles the gait is almost purely spastic. Only when paresis of the legs ensues does the gait become more dragging and labored, with slow, stiff steps ("spastic-paretic gait"). The ataxia is then, of course, no longer very noticeable.

The spastic-ataxic phenomena are generally equally well pronounced in both legs, but occasionally the disturbances may be more pronounced and exist for a longer time in one leg than in the other. We have already referred to the early ataxia of the trunk which is frequently found. In the arm the disturbance is often very slight for a long time, so that the patients themselves are scarcely aware of it. However, if careful tests be made (touching a fixed point with the index finger), slight disturbances are generally demonstrable. But, as a rule, the ataxia or the true intention tremor (*vide supra*) of the arms becomes marked fairly early, and may, as has already been mentioned, occur in its most pronounced form. The tendon reflexes of the upper extremities are also often increased.

In two other muscular regions the motor disturbances may give rise to very striking symptoms—in the muscles of articulation (tongue) and the muscles of the eye. Charcot laid great stress on these disturbances, and, in fact, in many severe cases they are very pronounced. It must be emphasized, however, that they are also frequently absent or only slightly developed. The disturbance of the muscles of the tongue and the other speech muscles is occasionally evident on merely protruding the tongue. It is most noticeable, however, in talking. Many patients with multiple sclerosis show a very characteristic disturbance of speech. The speech is slow, monotonous, scanning, indistinct, and sometimes finally almost unintelligible. The patient sometimes pronounces almost every syllable, or even every sound, separately, thus: "S-w-al-low." We sometimes notice that the voice often breaks into a falsetto. There can hardly be a doubt that this disturbance of speech indicates the presence of sclerotic nodules in the pons and medulla, and that the anomaly of motion at the bottom of it is analogous to the ataxia of the arms and legs described above. The nystagmus sometimes seen in multiple sclerosis, especially when looking to one side, is also entirely analogous to the other motor disturbances. But we must confess that we have seen manifest nystagmus in multiple sclerosis only in a comparatively small number of cases.

It is not easy to explain the occasion of all these motor symptoms of multiple sclerosis thus far described, but we may certainly say this—that the persistence of the axis cylinders in the sclerotic nodules mentioned above explains the fact that there is so often in multiple sclerosis not a real paralysis, but only ataxia (intention tremor) and spastic symptoms (increased tendon reflexes). The general tendency is to refer the ataxia to nodules in the posterior columns or in the superior lemniscus, and the spastic symptoms to nodules in the lateral tracts. The occasion of the oscillatory tremor is still wholly unknown. In my opinion, it is due to a disturbance of the mutual reflex relations of the antagonists to each other.

There are still, however, some other symptoms in addition to those already described which must be enumerated. They are not prominent symptoms in the general clinical picture, but are nevertheless of the greatest diagnostic importance. The cutaneous reflexes deserve the first consideration. Whereas, the tendon reflexes are almost invariably increased, we often find a marked diminution of the cutaneous reflexes. The plantar reflex is often very slight, but much more remarkable is the loss of certain other reflexes—namely, the cremaster reflex and, above all, the abdominal reflex. Since my attention has been called to this fact, I have found it confirmed almost without exception in more than one hundred cases. The loss of the abdominal reflex occurs very early. It is of great importance, as this disease generally occurs in young people with tense abdominal muscles, where the reflexes are normally hardly ever entirely absent. A second extremely important symptom is the pallor of the temporal half of the optic disc, which can be frequently demonstrated by ophthalmoscopic examination (Uhthoff and others). This objective symptom often occurs very early, though the patients themselves are not aware of any marked visual disturbance. Complete atrophy and blindness occur finally in a comparatively few rare cases. On the other hand, however, the contrast between the marked ophthalmoscopic changes and the comparatively insignificant disturbance of vision which is occasionally found is remarkable. This condition is probably due to the conservation of the axis cylinders. Nevertheless, on careful examination, we find not infrequently a contraction and limitation of the visual field, disturbances of color vision, etc. It is very curious that occasionally, at the onset of the disease (*vide infra*), total blindness of one eye occurs for a short time, and then disappears again. In rare cases optic neuritis and choked disc have been observed, which are due perhaps to retrobulbar sclerotic foci. Disturbances of the ocular muscles associated with double vision not infrequently occur, occasionally even as the initial symptom. The ocular palsies are either unilateral or bilateral. Occasionally we find paralysis of associated movements (foci in the pons). Loss of pupillary reflex

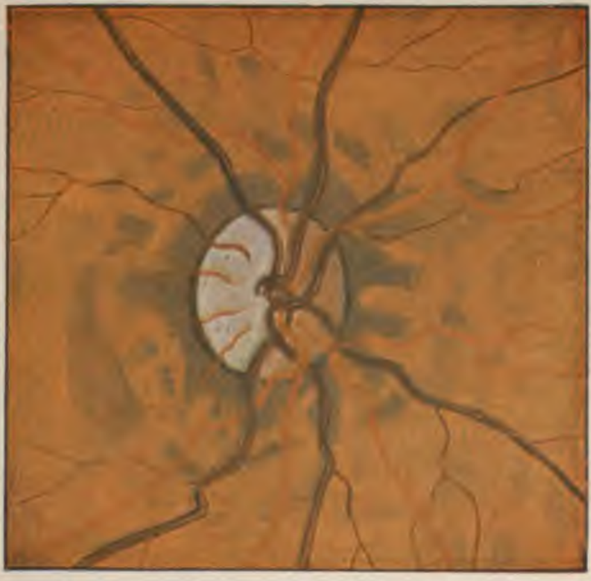

FIG. 159.—Pallor of the temporal half of the optic nerve (beginning optic atrophy in multiple sclerosis).

to light is scarcely ever observed. Disturbances of hearing are much rarer, but I have also repeatedly observed them.

Sensory disturbances are hardly ever pronounced in the clinical picture of multiple sclerosis. Upon accurate examination, however, slight and transitory hyperæsthesias cannot infrequently be demonstrated. Marked pain, also, does not belong to the typical clinical picture, but anæsthesia and painful sensations frequently occur. Severe and even neuralgic pains occur only in a few cases; occasionally they may appear as initial symptoms. Marked vesical disturbances are also rare, and generally occur only in the terminal stage of the disease. Slight vesical disturbances (transitory difficulty in urination, slight incontinence) can, however, be frequently demonstrated on more careful investigation.

Of cerebral symptoms we have yet to mention headache and especially vertigo. The latter is often a very early symptom of the disease. It may be paroxysmal or persistent. In the course of the disease there often appears a certain mental weakness, an imbecility, which sometimes increases to marked dementia. Conditions of melancholia or exaltation are much rarer. We must also mention the occurrence of apoplectiform attacks as a rare symptom, which is, however, often important in diagnosis. After slight prodromal symptoms, such as headache and vertigo, loss of consciousness and hemiplegia come on quite suddenly. With this the face is usually red, the pulse is frequent, and the temperature may rise to 104° or 107° F. (40° to 42° C.). After a day or two the consciousness gradually returns, and the hemiplegia soon disappears. Epileptiform attacks are much rarer. We saw these repeatedly in a typical case; they were mainly unilateral, and were followed by a hemiplegia which soon passed away. The precise cause of these attacks is still wholly unknown. We know only that they are not due to complicating gross changes in the brain, but that they arise merely from functional conditions of paralysis or irritation. Finally, we must mention an interesting symptom frequently observed in multiple sclerosis, and therefore of value in diagnosis—namely, the so-called impulsive laughing and crying. In many patients these emotional manifestations occur very frequently and in an uncontrollable fashion, either almost spontaneously or especially upon the slightest occasion. Not infrequently the two manifestations pass almost directly into each other.

In regard to the general course of typical cases, the disease develops very slowly and gradually. The more accurately we inquire into the history of the case, the more frequently we will be able to trace back the first indications of this approaching serious disease to a very remote period. These transitory premonitory symptoms are most remarkable, their occurrence is learned only by a careful history, and their importance, as a rule, becomes clear only very much later when the clinical picture of multiple sclerosis is already fully developed. Among the premonitory symptoms which often precede the onset of the continuous symptoms by many years, are, first of all, brief visual disturbances, occasionally lasting only a few days (almost total blindness of one or both eyes); furthermore, transitory weakness of one arm or leg, or sometimes of the entire side of the body. Neuralgic pains, etc., occur much more rarely. If marked and continuous symptoms occur later on they are confined at first generally to motor disturbances. Motor symptoms, tremor, uncertainty of movements, and disturbances in gait, usually appear in the extremities first. The patient often complains at the same time of occasional headache and vertigo. The speech gradually becomes more indistinct, the intelligence weaker, and the other symptoms of the disease described above develop. The affection almost always lasts for years and years. Variations in the course, temporary improvement, and new relapses are common. It would even seem in mild cases as if there were sometimes a recovery or at least an arrest of the symptoms, but in general we must always regard the prognosis as very unfavorable. We often see the condition rapidly grow worse, especially after the above-mentioned apoplectiform attacks. The last stage is characterized by the gradually increasing disturbance of the general nutrition, and, finally, by paralysis and bedsores. Death ensues from intercurrent diseases, from the increasing weakness, or sometimes in an apoplectiform attack.

UNUSUAL FORMS.—Besides the typical form of multiple sclerosis described, there are often, as we have said, cases that vary from the type (*"formes frustes"*). We will mention briefly the following possibilities:

1. The disease may be very latent. We saw one case in which, for a long time, the only symptom was a complaint of slight headache and vertigo. Finally, there was a transitory apoplectiform attack, several months later an epileptiform attack, and a few days after that death took place. The autopsy showed a completely developed multiple sclerosis.

2. Sometimes the disease appears under the exact type of a chronic transverse myelitis. The cerebral nodules cause no symptoms, they are present, perhaps, only in small numbers, and the spinal nodules cause a gradually increasing paraplegia of the legs, with vesical disturbance, loss of sensibility, etc. We have notes of several autopsies of multiple sclerosis in which, during life, the diagnosis of a simple transverse myelitis or a tumor had been made. The differential diagnosis is possible only by taking into consideration the entire course of the disease and the symptoms (examination of the eyes!).

3. Cases have been repeatedly known where multiple sclerosis has appeared under almost the exact type of a spastic spinal paralysis (*vide infra*). In these cases many nodules were situated in the lateral columns of the cord. If the spastic symptoms be combined with muscular atrophy (nodules in the anterior gray cornua), the disease may even simulate the type of an amyotrophic lateral sclerosis, with at times coexisting bulbar symptoms (*vide infra*). If multiple sclerosis be localized to an unusual extent in the pons and medulla, the symptoms of a chronic bulbar paralysis may be prominent.

4. Much less frequently the type of the disease may resemble tabes (immobile pupils, ataxia, pain). We do not know whether absence of the patellar reflex has been seen in undoubted multiple sclerosis. There are, however, combinations of multiple sclerosis and gray degeneration of the posterior columns (syphilis?).

5. It sometimes happens that multiple sclerosis is the reason for a slowly developing hemiplegia, which may then be falsely regarded as cerebral, while the autopsy shows several nodules in the corresponding side of the cord and pons.

6. In some cases the mental disturbances (dementia) are so prominent that there is the pronounced picture of paralytic dementia with disturbances of speech, etc.

Diagnosis.—In all such atypical cases the diagnosis of multiple sclerosis is sometimes quite impossible, or at best it can be made with a fair amount of probability only when some, at least, of the characteristic symptoms of the disease are present besides the anomalous symptoms. The circumstance, indeed, that the anomalous cases will not properly fit the molds of any other form of disease, should make us think of the possibility of a multiple sclerosis, and prompt us to look for characteristic symptoms. We would again emphasize the especial importance of the premonitory symptoms previously mentioned, the ophthalmoscopic examination (temporal pallor), absence of the abdominal reflexes, the possible presence of forced laughter, etc. All these symptoms are also important in beginning and but little developed cases where there may be even doubt at first as to whether we are dealing with a true organic nervous disease or only with a functional "hysterical" or "anemic" disturbance. The presence of the Babinski [or Oppenheim] reflexes together with the absence of the abdominal reflex, is very important in diagnosticating beginning cases. A slight ataxic uncertainty of the arms in touching fixed points can often be demonstrated very early by careful examination.

In typical cases, however, the diagnosis is generally easy, and often it can be made with absolute certainty. The slow development of the disease, usually without any discoverable cause, the comparative youth of the patient, the ataxia of the legs and the ataxia or tremor of the arms, the swaying of the body when standing with the feet together, the uncertain, spastic-ataxic gait, the absence of the abdominal reflex, the Babinski [and Oppenheim] toe reflexes, the characteristic ophthalmoscopic changes of the optic nerve, the increased tendon reflexes in the arms and legs, the absence of any marked disturbances of sensation or of the bladder, in some cases also the disturbance of speech, the nystagmus, the impulsive laughing and crying, the slight mental weakness which often comes on later, and, finally, the occasional apoplectiform or epileptiform attacks—all these symptoms give a very characteristic picture, which is usually quickly recognized and correctly interpreted. It is hardly possible to confuse it with paralysis agitans (*q. v.*) if we remember the characteristic attitude of the latter affection, the oscillatory tremor of the fingers when at rest, the rigidity of the face, etc. It is very seldom difficult to distinguish it from true tabes dorsalis. It is easier to confuse multiple sclerosis with Friedreich's hereditary ataxia (*q. v.*); but the condition of the tendon reflexes is usually decisive; these are wholly absent in tabes and Friedreich's disease, but they are almost invariably increased in multiple sclerosis. It sometimes happens that hysteria assumes a form very similar to multiple sclerosis (tremor, ataxia, increased tendon reflexes, etc.), but in such cases we can usually arrive at a correct diagnosis by careful observation, if we pay attention to the whole condition of the patient, the course of the disease, and the characteristic hysterical "stigmata." It is certain that beginning cases of multiple sclerosis were formerly frequently looked upon as hysteria. Especially the transitory and often apparently severe premonitory symptoms (for example, the transitory almost total blindness, without any ophthalmoscopic changes), were frequently interpreted as hysterical symptoms. There is, however, a combination of multiple sclerosis and hysteria (especially hysterical hemianæsthesia), as has been maintained especially by Charcot's pupils. The distinction between multiple sclerosis and the so-called pseudo-sclerosis will be stated below.

Prognosis.—The most important features in prognosis have already been mentioned. We have seen that an improvement in the symptoms is not uncommon, but that the final outcome of the disease, with perhaps a few exceptions, is unfavorable. The disease may last for many years.

Treatment.—In the treatment we may make a trial of the same remedies that have been mentioned in the description of myelitis. Absolute rest and good care, which many patients can obtain only in a good sanitarium, are often beneficial. We may also use galvanism, baths (half baths, carbonic-acid baths, electric baths), and sponging, and internally iodid of potassium, ergotin, nitrate of silver, small doses of aspirin, etc. Recently some physicians claim to have obtained remarkably good results from injections of fibrolysin. Personally, I have not yet had any experience with this treatment. The uncertainty of movement can sometimes be distinctly improved by methodical gymnastic exercises (see the following chapter).

APPENDIX

THE SO-CALLED PSEUDO-SCLEROSIS

Cases of disease have been observed, first by C. Westphal and later by the author and others, the symptoms of which seemed to warrant the diagnosis of multiple sclerosis, but the autopsy proved wholly negative. The victims are usually young and often have a predisposition to nervous disease. The symptoms of the disease consist of a marked rapid or slow oscillatory tremor with wide excursions, which sometimes occurs during rest, but which becomes unusually violent on motion. The tremor affects the arms particularly, but also the trunk and legs. There seems to be no real ataxia as in multiple sclerosis. The tendon reflexes are increased and a distinct spastic rigidity may devélop in the legs. The speech is sometimes quite as decidedly scanning as in multiple sclerosis. Nystagmus is usually absent. We have seen apoplectiform attacks in a pronounced form, and also impulsive laughing and crying. The face often has a peculiar rigid expression. Certain mental disturbances are very characteristic, especially a marked tendency to outbursts of wrath and actual maniacal attacks. Signs of confusion and hallucinations are less common. The course may extend over many years. The disease seems to be incurable. Death ensues through increasing general weakness or in an apoplectiform attack. Anatomical investigation, as has been said, is essentially entirely negative, even with a careful microscopical examination of the nervous system.

The resemblance between the type of this "pseudo-sclerosis" and true multiple sclerosis is evident; nevertheless, now that we know the disease, we can often make the distinction. The chief points to bear in mind are the very marked true oscillatory tremor, the absence of real ataxia, and the mental disturbances just mentioned.

The treatment of pseudo-sclerosis is the same as that of multiple sclerosis.

CHAPTER VII

TABES DORSALIS

(*Locomotor Ataxia. Gray Degeneration of the Posterior Columns. Ataxie locomotrice progressive.*)

We give at present the old name of tabes dorsalis, "consumption of the spinal cord," to a perfectly definite chronic disease of the central nervous system, whose chief, but by no means sole, anatomical basis is regarded as a primary systemic degeneration of the peripheral sensory neurons, both of their peripheral branches, and more especially of their processes in the posterior columns of the spinal cord. The first description of tabes, which, of course, is defective in many respects, is found in a work of W. Horn in 1827. We must thank especially the investigations of Romberg in Germany, in 1851, and of Duchenne in France, in 1858, for a more comprehensive knowledge of the disease and for a precise distinction between it and the other chronic diseases of the spinal cord. From then up to very recent times, tabes has been the subject of numberless investigations; but it always offers new phases and new problems to investigators.

Ætiology.—But little that is definite was known until lately as to the causes of tabes dorsalis. Hereditary conditions play a very slight part in genuine cases, and even a general "neuropathic taint" can only rarely be made out in ataxic patients. Much weight in regard to ætiology was formerly laid upon previous exposure to cold. It cannot be denied that sometimes the first symptoms of the disease follow some pronounced exposure to wet or cold, but much more frequently nothing of the sort can be made out. The case is similar with regard to physical and mental overexertion, which were formerly made answerable for the origin of tabes. It is an utterly ungrounded assertion that sexual excesses may be the cause of tabes. Tabes may rarely develop as a result of acute diseases or somewhat more frequently as a result of injury (general concussion of the body from a fall, fractures, etc.); but we may ascribe to these factors merely the significance of an exciting cause. In these rare cases, too, it is hard to confirm the connection. The earlier teaching that tabes develops after "suppression of the foot sweat" is manifestly due to a confusion of cause and effect. The absence of foot sweat is not the cause, but a symptom of incipient tabes.

The only ætiological fact which in my opinion is beyond any doubt is the relation of tabes to a previous syphilitic infection. This relation of the two diseases to each other was first confirmed in France by Fournier and in Germany by Erb, and in spite of the vigorous contradiction which the views of these observers at first met with on many sides, this theory of tabes as a disease having a direct ætiological relationship to syphilis is constantly acquiring more and more general recognition.

The connection between tabes and syphilis can be confirmed, first of all, by statistics. Erb was able to find a history of syphilis, with secondary symptoms, in about sixty-two per cent of his patients; and Fournier, in 103 cases, found syphilitic antecedents as many as 94 times. Our own observations agree exactly with Erb's data, since sixty-one per cent of our patients stated definitely that they had formerly suffered from syphilis. If we also reckon the cases where the patients admit a former sore but no secondary symptoms, the percentage becomes much greater—ninety per cent. It is worthy of note that in most cases of tabes the previous syphilis has not had a great intensity. In the histories of many of the cases of tabes we find, therefore, merely a statement about a "small ulcer of the penis," or that specific treatment had been used for a short time "as a precaution," etc. It is noticeable that in a case of tabes one can scarcely ever regard the assumption of a previous syphilis as entirely improbable, or even as impossible. The search for the "tabetic virgin" has remained fruitless, in spite of all our efforts. Only quite infrequently do we find tertiary syphilitic symptoms as well as tabes; we have seen, for example, severe ulcers of the skin, gummatous periostitis, etc. The time between the infection and the beginning of the first symptoms of tabes varies very much; it may be from two to twenty years.

The interesting serological and cytological examinations of the last few years have furnished a new series of valuable proofs for the ætiological relationship between tabes and syphilis. In the numerous examinations which have been conducted, the Wasserman test for demonstrating a previous syphilis by complement deviation has given a positive result in the majority of cases of tabes, and, as we shall see later on, in the closely related general paralysis. In typical general paralysis the reaction is almost invariably positive with the blood serum; it is not quite as constant, however, for tabes, though at any rate it is found in seventy to ninety per cent of the cases. With the cerebro-spinal fluid the Wasserman reaction, as we may note here, is found to be positive almost as regularly as with the blood serum in general paralysis; while, according to the most recent reports of Nonne, it is only exceptionally found to be positive in tabes. The cytological and chemical changes of the cerebro-spinal fluid, which are important for diagnosis, will be referred to more in detail in the discussion of the symptomatology of tabes.

The connection between tabes and previous syphilis also explains naturally a number of peculiarities in the occurrence of tabes, especially the fact that tabes is much commoner in men than in women; that it is particularly very rare in women of the better class, while it is found much more frequently in women of the lower classes; that it comes on especially in middle life; that it is much more common in the higher classes and in urban populations than in the country; that certain classes (merchants, artists, officers) show a much larger percentage of cases of the disease than others (clergymen, teachers, etc.). If we see, however, any cases of tabes which show striking differences in these respects, we can almost always find evidence, even in such cases, of a previous syphilitic infection. Such individual cases, therefore, afford material for the most convincing proof of the connection between the two diseases. Thus, we sometimes see tabes in perfectly irreproachable women; here we can almost always detect previous syphilis in the husband which has been transmitted to the wife. We ourselves and other observers have seen tabes arise in this way in both husband and wife. We may also find tabes in very young or very old persons, and then we can discover that syphilis was acquired at a very early or a very advanced age. A few cases of tabes occur in children, and are in all probability to be referred to hereditary or infantile syphilis.

Although the connection between tabes and previous syphilis may be regarded as definitely established, the nature of this connection, on the other hand, is not yet fully explained. This much, of course, is certain, that tabes is not an ordinary tertiary syphilitic disease. Both its anatomical changes (*vide infra*) and the extremely slight effect of antisyphilitic treatment remove every doubt as to this. The connection between the two diseases must be of some other nature, and therefore we long ago expressed the opinion that tabes was due only to certain chemical poisons (toxins), which develop in the body from the action of the syphilitic infection. Such toxins may very well cause definite systems of fibers (*vide infra*) to degenerate, and their presence is also a good explanation of certain clinical peculiarities of tabes, especially the paroxysmal occurrence of certain symptoms (the so-called crises). Many of these crises, especially the gastric and intestinal crises, are very similar to toxic symptoms (vomiting, pain, pronounced general debility and weakness, etc.). Tabes is therefore a sequela of syphilis. It and the closely allied general paralysis therefore stand in the same relation to syphilis as " metasyphilitic nervous diseases," that the common " nervous sequelæ " do to the acute infectious diseases—for example, as postdiphtheritic paralysis and ataxia do to previous pharyngeal diphtheria. Of course this hypothesis does not afford a completely clear insight into the nature of the tabetic process. Thus, the constant progress of most (but of course not all) cases of tabes requires a further special explanation. We may perhaps imagine the process thus, that the toxins absorbed at first merely injure the affected fiber system (*vide infra*), but that the complete atrophy and cessation of function comes on gradually through the wear and tear associated with the normal functioning of the nervous elements. In this sense other external influences (fatigue and physical overexertion, exposure to cold, injury) may be regarded as aiding the process. In particular, a series of tabetic cases have been observed in which the symptoms developed after a severe trauma (fall on the back, etc.). We can readily imagine that the trauma first rendered the nerve tracts in question susceptible to the syphilitic toxins. Nevertheless, even in these cases of traumatic tabes, the preceding infection is the *conditio sine qua non*. As a rule, also, we are not dealing with the onset of the tabetic changes after a trauma, but only with the first appearance of more marked clinical symptoms.

No one will deny that a preceding syphilitic infection cannot be proved in every case of tabes. But the very same thing is true for undoubted syphilitic lesions, such as gummata of the liver, etc. The only consistent scientific point of view is to regard every true tabes as a sequel to a preceding syphilitic infection. At all events, it would not be impossible that other noxious influences might cause the same systems of fibers to degenerate as in tabes, and thus produce a form of disease similar to that of genuine tabes. Thus, we know at present, from the investigations of Tuczek, that symptoms may develop in chronic ergot poisoning (ergotism) which are perfectly analogous to those of tabes and depend upon a corresponding disease of the posterior columns of the cord, which can be demonstrated anatomically. These cases of " tabes," however, which are traced to some other cause, must be distinguished from the metasyphilitic cases which constitute by far the great majority. The question why only a small percentage of syphilitics acquire tabes later on can be as little answered as the questions why only a fractional part of the diphtheria cases are followed by paralysis, or why a nephritis occurs after only a small percentage of the scarlet-fever cases. We can think of differences in the individual disposition (neuropathic?) or of differences in the virus. French investigators assume that there is a particular "*syphilis à virus nerveux,*" an assumption without very much foundation, though some peculiar clinical experiences can be adduced in its favor. Thus, the occurrence of tabes or paresis in different individuals who acquired their infection from the same source, and, furthermore, the occurrence of tabes in married couples with the same initial symptoms, etc., have been noticed.

Pathological Anatomy.—As we have seen on page 152, the cell bodies of the peripheral sensory neurons lie in the spinal ganglia. The processes of the spinal ganglion cells divide into two branches. One goes to the periphery of the body, the cutaneous surface; it forms the so-called peripheral sensory nerve. The other process serves for the centripetal conduction of sensory irritations; it enters the cord through the posterior roots and goes, as we have said above, either upward in the posterior columns or into the posterior gray cornua, there to connect with spinal sensory neurons of the second order. Upon this peripheral (first) sensory neuron system (spinal ganglion system), whose anatomical arrangement we have just briefly described, the (toxic) cause of tabes acts especially and causes it slowly, fiber by fiber or neuron by neuron, to degenerate. In tabes, as in most other primary systemic diseases, we find that the evident visible anatomical degeneration of the different neurons always begins in the prolongations, and the destruction of the cell body itself follows later, if at all, and is the last process. Since the prolongations of the spinal ganglion cells are, first, the peripheral sensory nerves, and, second, the fibers in the posterior columns, and, in part, in the posterior horns of the spinal cord, we understand, without further explanation, that the anatomical process in tabes must manifest itself as a degeneration both in the peripheral nerves and in the posterior columns and posterior horns of the spinal cord. So far as we can at present appreciate the conditions, the spinal process of the neuron cells in the spinal ganglia seem to be affected earlier, more regularly, and more extensively than the peripheral process; but it seems to us probable that there are individual differences in this respect, and that many differences in the course of the disease in different cases of tabes may be explained by such variations in the particular anatomical localization of the tabetic degeneration.

If we examine the spinal cord of a patient who has died in the advanced stage of tabes, the smallness and thinness of the cord usually strikes us at once. The pia mater is thickened and opaque, especially on the posterior surface. We often see the posterior columns appearing through the pia as a gray band extending the whole length of the spinal cord. On cross section we notice that the smallness of the cord is due chiefly to the atrophy of the posterior columns, which is often very considerable. These no longer protrude backward, as they normally do, but seem flat and sunken. From their pronounced gray color they are very plainly distinguished on cross section from the rest of the white matter of the cord. The posterior cornua of the gray matter, and the posterior nerve roots, show exceptionally a considerable atrophy, and appear very small and thin and also of a gray color.

Microscopic examination gives more exact information as to the extent and form of the degeneration. This shows (Fig. 160, *a, b, c*) that all portions of the posterior columns are not affected in like manner, but only those portions which contain the processes and collaterals of the posterior root fibers, which therefore belong to the system of spinal ganglion neurons. The other tracts (short tracts, commissural fibers) are spared. The degeneration is almost always most intense in the lumbar cord; here it affects chiefly the middle and posterior portions of the posterior columns, while the most anterior portion and the so-called dorso-median sacral bundle (Fig. 160, *c*) remain normal in almost all cases. There are indeed fibers which have another origin and do not belong to the spinal ganglion system. In the dorsal cord (Fig. 160, *b*) the posterior columns are almost completely degenerated. There are usually small normal areas still preserved in the posterior external and the most anterior portions. In the cervical cord (see Fig. 160, *a*) the so-called columns of Goll are chiefly affected, together with the prolongation of the fibers from the root zones of the lumbar cord, and also the "lateral root areas"—that is, those portions in the columns of Burdach where fibers enter directly from the posterior nerve roots, and from which fibers may be traced farther into the gray matter of the posterior cornua; but the so-called posterior external areas, and also two little antero-lateral areas, remain entirely, or at least for a long time, free from the disease, because there are also bundles of fibers which do not belong to the system of the spinal ganglia or posterior roots. If we

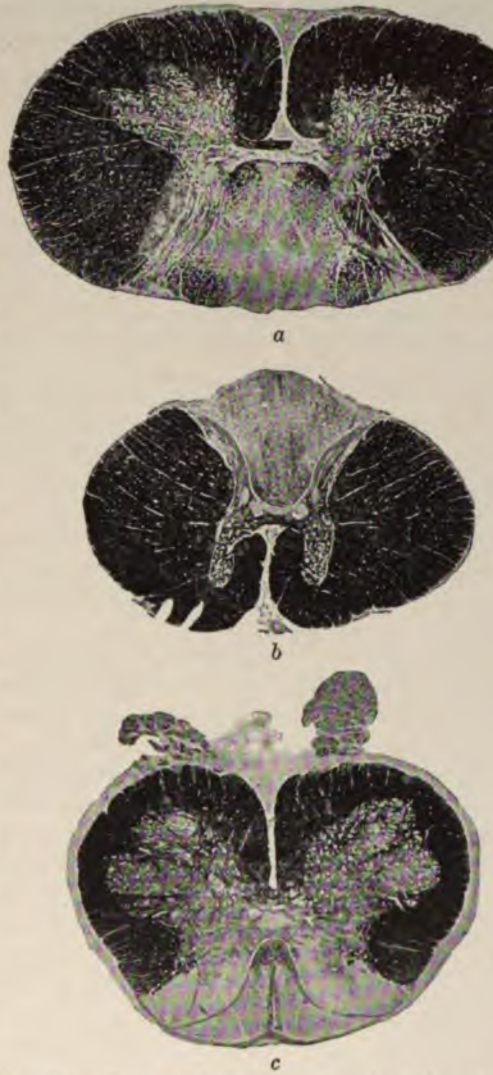

FIG. 160.—Degeneration of the posterior columns in advanced tabes. *a*. Cervical cord. *b*. Dorsal cord. *c*. Lumbar cord. (Personal observation.)

examine cases of tabes in the earlier stages which happen to come to autopsy, we find in the posterior columns only a few root territories affected—that is, the fibers arising from individual spinal ganglia or posterior roots and lying near one another, while other root territories remain free from degeneration (Figs. 161 and 162). A system of very fine fibers, entering through the posterior roots, is affected very early, as a rule. It branches outward immediately after the entrance of the roots, and occupies a small but very sharply defined territory at the point of the posterior cornua, between the posterior and lateral columns (the so-called zone of Lissauer). In all the affected root territories the degeneration slowly involves fiber after fiber. The order in which the different root territories degenerate one after another and in part at the same time with one another, is certainly not always the same in individual cases, as is evident from the variations in the clinical course of the disease. As a rule, the neurons of the lumbar and lower dorsal spinal ganglia degenerate first, and the neuron systems of the cervical ganglia later.

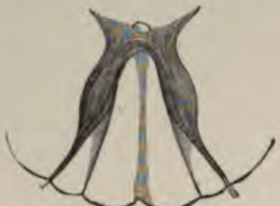
FIG. 161.—Dorsal region.

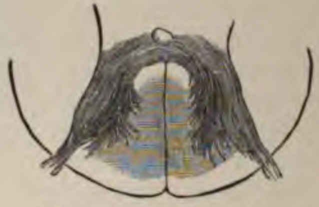
FIG. 162.—Lumbar region.

Transverse section through the posterior columns of the cord in beginning tabes dorsalis.

We must state, with reference to the participation of the gray matter in the disease, that the posterior cornua, as we have already said, are also found considerably affected, which is explained mainly by the atrophy of the posterior root fibers which enter them directly. It also cannot appear strange that the medullated fibers found in Clarke's columns seem very much reduced in number, since they also are direct processes of the posterior root fibers. The cells of the columns of Clarke remain normal. The ganglion cells of the posterior horns, which belong to the second sensory neurons, also remain normal, and no changes are found in the processes of these cells in the lateral columns. The lateral and anterior columns of the cord usually remain unaltered (*vide infra*).

On the other hand, the peripheral sensory nerves—that is, peripheral processes of the spinal ganglion cells—are by no means spared. In all cases of tabes which have been carefully examined thus far, we find in the peripheral nerve trunks (sciatic, crural, etc.), and usually still more in the finer branches of the sensory nerves, a considerable number of degenerated centripetal fibers (Dejerine, Oppenheim and Siemerling, and others). We regard this degeneration as precisely analogous to the disease of the posterior columns and by no means a secondary degeneration. Like the degeneration of the posterior columns, it also is an expression of the primary disease of the peripheral sensory neuron system. In regard to the cell bodies in the spinal ganglia, those certainly are well preserved for a long time, at least in regard to their external aspect, but finally we may find more or less definite changes in them, although hardly ever complete atrophy.

The anatomical process of tabes is by no means limited exclusively to a slow degeneration of the first sensory neuron system. Other neurons and neuron systems may also be involved in the disease. The occasional affection of the trigeminal neurons is, of course, wholly analogous to the degeneration of the sensory spinal neurons; but other regions sometimes become degenerated, especially the optic nerve, more rarely the vagus and acoustic, and also certain motor regions, especially the ocular nerves, other peripheral motor nerves, the pyramidal lateral tract, etc. We will return more fully to these "complications," or, to speak more correctly, to these rarer localizations of the tabetic process, in the clinical portion of this chapter. If we consider, however, that the whole neuron territory of the spinal ganglia evidently contains neurons of very different physiological importance (fibers for the conduction of the different qualities of sensation, for the coördination of movement, for the innervation of the bladder and rectum, for reflex processes, etc.), and that also other most diverse neuron territories may be involved in tabes, we can understand that tabes can be termed, not a simple, but far more properly a "*combined systemic disease*."

The form of the tabetic affection, in my opinion, consists of a primary degenerative atrophy of the nerve fibers, and a corresponding secondary increase of the connective tissue. The gray color of the posterior columns is due to the loss of the medullary sheaths. Since the destruction of the nerve fibers advances but very slowly, we find only a few fatty granular cells. In old cases we find numerous corpora amylacea, whose origin and significance are still unknown. The thickening of the pia mater is a purely secondary phenomenon, due to the inflammatory irritation by the products of decomposition of the degenerating nerve fibers. The lymphocytosis of the cerebro-spinal fluid is also due to this cause (*vide infra*). The associated "chronic meningitis" is always most marked on the posterior surface of the periphery of the cord, corresponding to the atrophy of the posterior columns. A number of investigators are still inclined to attribute the entire tabetic process to a primary (syphilitic) meningitis. Nageotte, especially, has expressed the opinion that a primary leptomeningitis, characterized by specific syphilitic changes in the small vessels, produces primarily a neuritis of the posterior roots, and later on leads to secondary ascending degeneration of the posterior columns. How this theory can find any adherents is inexplicable to me. Why should a primary meningitis always develop only on the posterior surface of the spinal cord? Why is it that ninety per cent of all the cases are associated at the outset with a loss of pupillary reflex to light? These and other objections, in my opinion, militate very strongly against the theory of a primary meningeal origin for tabes. On the other hand, I admit frankly that occasionally true syphilitic lesions of the meninges may be found associated with the specific tabetic degeneration, which is not at all surprising, in view of the fact that a syphilitic infection is always present.

Clinical History.—A disease which has as its basis so definite and strictly limited an anatomical change as is the case with tabes dorsalis would also be expected to give a very characteristic clinical picture. This supposition is entirely correct, and there are few diseases which can be diagnosticated, even in their earliest stages, with as much certainty as tabes. This fact is explained only by regarding tabes as a systemic disease, in which certain systems of fibers are always affected, while others are as constantly spared by the disease. The difference between individual cases of tabes lies, therefore, less in the symptoms themselves than in their intensity, their duration, and the order of their occurrence. In this regard, however, the differences in the clinical pictures are extremely varied, so that, even with a comparatively great personal experience, we often see new combinations of symptoms and also peculiarities in their course.

For the majority of cases we may sketch the following general description of the disease, in which it is better to divide the whole course into several stages; but, of course, this division can have only a schematic value.

Tabes begins, as a rule, with a stage of initial symptoms, which develop very gradually and insidiously, and which may be of very varying duration. The most characteristic symptoms of this stage are those of sensory irritation, most frequently in the form of the so-called lightning-like, "lancinating" pains in the lower extremities. They are sometimes very severe, but at other times only of slight intensity, and are comparatively little noticed by the patient, who regards them as "rheumatism." Many patients have a feeling of numbness and tingling in the tips of the fingers, especially of the ring and little fingers, and there is often a pronounced girdle sensation in the trunk. In some cases, too, neuralgic and migraine-like pains in the head may appear in the early stages.

Besides these symptoms of sensory irritation, which may often be for years the only symptoms of which the patient complains, two objective symptoms appear very early, which are of the greatest importance in the diagnosis of incipient tabes—the disappearance of the tendon reflexes of the lower extremities (patellar and Achilles reflexes) and the reflex immobility of the pupils. The absence of the Achilles and patellar reflex is the most constant of all the known symptoms of tabes (*vide infra*). The reflex immobility of the pupil—that is, the failure of the pupil to contract to light, while the changes on accommodation may be perfectly retained—is, indeed, not so constant as the failure of the patellar reflex, but it is present in almost all cases. In my experience, at least, it appears, as a rule, earlier than the loss of the tendon reflexes. If all three symptoms—lancinating pains, absence of patellar reflex, and immobility of the pupils—are present at the same time, the diagnosis of tabes is absolutely certain, even if all other symptoms are wanting, because this peculiar combination of three such apparently heterogeneous symptoms is seen in this disease alone. If only two of these symptoms are present, but if the reflex immobility of the pupils be one, the diagnosis is also practically certain; but if this symptom be absent we should be very cautious in admitting that the affection is genuine tabes.

Among the rarer initial symptoms we shall also learn to recognize diplopia, caused by paralysis of certain ocular muscles, and loss of vision, from optic atrophy. In many other cases certain disturbances of cutaneous sensibility (a girdle feeling, paræsthesia, a furry feeling) announce the approach of the disease. Sometimes disturbances in micturition appear quite early, while in other cases gastric crises (*vide infra*) or some joint affection are the first symptoms which the patient notices.

After this first stage of the disease has lasted for a very varying period, from a few months to two, or five, or even twenty years, the second stage begins; this we usually term the ataxic stage of tabes.

The beginning of this stage is recognized by the appearance of disturbances of gait. The gait becomes more difficult and more uncertain, and there are certain peculiarities which we will describe more fully later. Careful examination shows that the disturbance in gait is due not to a paresis of the muscles but to a disturbance of coördination, ataxia of the lower extremities. This symptom usually increases very slowly, until it reaches a degree where the patient can walk only with assistance, and finally cannot walk at all. There is often later, but almost always not for years, ataxia of the upper extremities.

Besides the persisting symptoms of the first stage, there are often now more marked disturbances of sensibility, as well as ataxia. The patient has a feeling as if he were walking on wool, felt, or similar substances. If he closes his eyes there is great swaying of the whole body—"Romberg's symptom." Physical examination of the sensibility almost always shows considerable disturbance of the cutaneous sensibility, especially of sensibility to pain, or even of other forms of sensibility. A loss of muscular sense especially is very common. The disturbances of micturition, such as incontinence, gradually become more marked, and very often cystitis gradually develops. This stage also may last for years. Sometimes the disease seems to stand still, frequently even manifest improvement is seen, but then the condition becomes worse again.

The third stage, the terminal stage of the disease, develops if the patient has not previously succumbed to an intercurrent disease. The symptoms are the same as in most of the other chronic diseases of the spinal cord. The patient gradually becomes more and more wretched and helpless, and finally is confined almost wholly to his bed. The ataxia reaches an extreme degree, and finally there is sometimes an actual motor weakness or complete paralysis of the legs. In these cases, which are by no means frequent, we are correct in calling the third stage of tabes the "paralytic stage." A severe pyelo-cystitis usually develops, bedsores appear, and death finally frees the patient from his lamentable condition.

We must now complete this briefly sketched picture of the disease by a more careful description of the single symptoms.

1. Disturbances of Motility.—Disturbances of motility of tabetic origin depend, in almost every case, not on a lesion of the centrifugal motor nerves, but on a lesion of the centripetal sensory nerves. We are dealing with sensori-motor disturbances, principally of the kind produced by a loss of centripetal stimuli from the organs of motion themselves (muscles, tendons, joints). The disturbances caused in this way affect the coördination of movements (ataxia), reflexes (loss of deep reflexes), and the muscular tonus (hypotonia).

The most noticeable and most characteristic motor symptom of developed tabes dorsalis is the disturbance of coördination, the ataxia (see page 204). This is almost always seen in the lower extremities first. To investigate ataxia, we test the patient's ability to stand and walk, as well as the movements of each leg. For the latter purpose it is most convenient to ask the patient to touch the kneepan of one leg with the heel of the other foot ("knee-heel test"). We see then that the leg moved is often carried beyond the point designated several times before it reaches it. The ataxia is often noticeable, even in crossing one leg over the other, as the leg raised makes much too great and too "throwing" a movement.

The alteration of the gait is very characteristic—the ataxic gait, from which we can often perceive the patient's disease at the first glance. If the patient sits down and tries to get up again to walk, there is difficulty in rising. He separates his legs to find a firm point of support, he takes a stick to help himself if he can, and he often gets the proper balance to keep himself erect only after several attempts. The gait itself is straddling, and the legs are raised abnormally high and set down with a stamp. The direction of the gait, however, is, as a rule, well preserved, and the trunk (*vide infra*) is often but little involved in the swaying. This is the distinction between the ataxic gait of many tabetics (due to ataxia of the legs) and the staggering, wavering gait in cerebellar disease ("cerebellar ataxia" with ataxia of the legs and trunk), in multiple sclerosis, etc. If we have the patient turn rapidly or make a proper military "about face," the uncertainty of movement is still more marked, as it also is if the patient rises suddenly and begins to walk or makes a sudden halt when walking. These methods of testing are therefore especially suitable for ascertaining the first beginnings of ataxia. Very often the first uncertainty in the gait is noticed in going downstairs, because the patient cannot put his foot securely on the next step below. Going upstairs is at first far less affected because the perpendicular rise of the stairs prevents any excessive movement. If the ataxia reaches a higher degree, most tabid patients always walk with a stick and control the movements of their legs by keeping their eyes fixed on the floor as they walk. This control is necessary because the sensibility of the legs, especially the muscular sensibility, is diminished at the same time.

In more advanced cases the ataxia becomes apparent even on standing with the feet close together (static ataxia). We see continually in the muscles and tendons of the legs slight contractions which are necessary to maintain the constantly shifting equilibrium. These contractions become much more marked as soon as the patient stands with his eyes shut. There is then usually an evident swaying of the body, which is sometimes so great that the patient is in danger of falling. This phenomenon (Romberg's symptom), which can often be demonstrated very early in a rudimentary form, is due to the defective regulation of the muscular movements, which are necessary to preserve the equilibrium, as a result of the impaired sensibility of the muscles, joints, and, in part also, of the skin of the soles of the feet themselves. As in all morbid conditions, the body attempts to replace the loss of essential functions as much as possible by the vicarious actions of other organs. The disturbance of motility of tabetics is therefore diminished as much as possible by an increased control by the visual sense. Ataxic tabetics, therefore, always fix their eyes on the floor while standing or walking. According to the degree of ataxia, the result of this visual control of the movements varies. At any rate the ataxia and, consequently also, the standing with the feet closely approximated, become worse as soon as the ocular control is eliminated. Some cases of initial tabes can still stand fairly well with the eyes open, but commence to sway markedly as soon as they close their eyes. From a like reason it is much harder for most ataxic patients to walk in the dark than by daylight.

In many cases of tabes, especially when the disease is far advanced, the coördination of the maintenance of the position of the trunk is evidently disturbed. Such patients finally cannot sit up, since the trunk sways so much. The gait of tabid patients with great ataxia of the trunk is still more uncertain and also plainly staggering.

If ataxia of the upper extremities occur in the course of the disease, it is easily recognized if the patient tries to take hold of some definite object, such as his ear, or if he brings the tips of his two forefingers together from a certain distance, or if he tries to touch exactly with the tip of his finger the finger of the physician when held before him. The ataxia is, of course, also apparent in all fine complicated movements (eating, sewing, writing, tying a knot, drawing straight lines, etc.). The movements are irregular and uncertain, and the excursions are marked. In the arms also the anomaly in the movements is still greater with the eyes shut.

There has been much written and much argued formerly as to the cause of the ataxia in tabes dorsalis. The main reason why it is at present impossible to give an entirely satisfactory explanation of the occurrence of ataxia lies in the fact that we are not yet in a condition to know and to analyze with sufficient accuracy the process of normal coördination of motion (page 204); for manifestly every theory as to the causes of ataxia must begin with the processes involved in the coördination of normal movements. If we try to get a clear idea of this, the most essential point to bear in mind is that coördination of motion is not a congenital function, but a power of our organs of motion acquired by practice. Naturally the ability to acquire presupposes a suitable organization, with which, however, every normal child is endowed by heredity at birth. The movements of little children who are learning to walk are ataxic, and even in later life it often happens that we have to learn how to perform certain complicated and difficult movements (so-called manual dexterity, exercises of skill). We can get no other idea of this "learning how to coördinate" than that it takes place by the aid of the constant action of controlling and correcting impressions coming from the periphery—that is, centripetal—but we must bear in mind that these actions are mainly unconscious. The surer we become in the execution of the movements, the more the regulatory influence of the centripetal irritations falls into the background, without ever wholly disappearing. In these cases we must not consider, by any means, merely the irritations which are brought to the central organs from the skin of the parts moved; but we should consider, just as much or probably far more, those irritations which are due to the varying tension and position of the deeper parts, the muscles, the fasciæ, the ligaments, and the articular surfaces. Even the special organs of sense, particularly the eye, under some circumstances, assist materially in regulating motion.

A disturbance of coördination must, accordingly, take place when this constant regulation of our movements by centripetal irritations ceases. Since the anatomical lesion in tabes, as we have seen, consists almost exclusively of a degeneration of centripetal systems of fibers passing through the posterior roots, there can be no doubt in my opinion that tabetic ataxia is due to the degeneration of centripetal fibers regulating movements; but we must emphasize again the fact that these centripetal coördinating fibers must not be entirely identified without further proof with the centripetal fibers serving for conscious sensibility, for a parallelism between the degree of ataxia and the severity of the sensory disturbances (in the skin and in the muscles), although, of course, disturbances of coördination and disturbances of sensibility very often coexist in this disease, has not yet been demonstrated, and is, according to my experience, certainly not always present. There are, however, undoubtedly cases of tabes with marked ataxia of the legs in which we can find on examination only slight disturbances of the cutaneous and muscular sensibility. This is still more frequently the case in other diseases attended with ataxia. We may therefore very well imagine that ataxia is the consequence of a degeneration of those collaterals which go from the centripetal neurons to the motor ganglion cells, and there provide for the regulation of the strength of motor innervation, although there are still enough conducting paths to conduct sensory impressions to the centers of consciousness. We do not as yet know, however, in what part of the motor tract the regulatory influence of centripetal irritations takes place—whether in the cells of the anterior horns of the cord, or higher up in the motor cortex, or in part in the cerebellum, etc. This can be made perfectly clear only by further investigation. We can already surmise that the normal coördination of our movements requires a fairly complex apparatus, and that therefore regulating centripetal influences must come into play at each individual motor station. In my opinion, the chief regulation of all complicated grasping and occupation movements occurs in the motor cerebral cortex, which is undoubtedly the termination of some centripetal lemniscus tracts. For this reason interruption of the lemniscus fibers high up and diseases of the motor cortex of the brain also produce ataxia. The important function of maintaining the equilibrium of the body in standing and walking probably belongs to the cerebellum, and it is undoubtedly a very important circumstance that the posterior root fibers enter the cells of Clarke's column and communicate farther on with the cerebellum by means of the lateral cerebellar tracts. The simplest coördinated relations of antagonistic groups of muscles to each other is perhaps performed by the regulation of the spinal motor ganglion cells through "reflex collaterals" reaching them from the posterior root fibers. The coördination of all our movements, therefore, does not follow a simple scheme, but depends on the amount and nature of the required movement. The motor innervation is subject to the continuous regulating influence of centripetal stimuli, either retarding or accelerating, just the same as well-trained carriage horses are constantly controlled with the reins. As soon as the reins are held loosely the movements become less certain, and every loss of centripetal stimuli to the motor centers, which can no longer be compensated for, necessarily produces a disturbance of motor innervation which is manifested as ataxia of movement.

Ataxia is therefore the specific motor disturbance in tabes. The crude strength of the muscles may be perfectly normal, and it is chiefly Duchenne's service to have made clear for the first time the principal distinction between ataxia and paralysis. He showed that ataxics, who can no longer walk a step alone, can nevertheless exert the greatest strength with their legs. We have ourselves treated a teacher of gymnastics who, in spite of the most marked ataxia of the arms, had still so much strength in them that he could support himself in bed on his arms and keep his whole body, with his legs extended, in the air. It sometimes happens, however, that the crude strength also disappears in tabes, and that the muscles become paretic. There may be a simulation of a certain "pseudo-paresis," since the patient with severe ataxia loses the ability to concentrate his muscular strength in the necessary direction. There is also in tabes occasionally, as we have said, an actual paralysis of the legs ultimately. We have stated above that even a complete paraplegia may finally develop in the course of the disease. In these cases we find, on anatomical examination, that the process is no longer confined to the posterior columns, but that there is also a systemic degeneration of the lateral pyramidal motor tracts in the lumbar cord. In some cases paraplegia has been observed to come on quite suddenly even in the early stages of the disease, and usually to disappear again in the course of a few weeks. Its origin has not yet been explained. The rare paralyses of certain motor nerves have another significance. They have usually been noted at the beginning of the disease, and affect the radial, peroneal, accessory, etc. They are probably due to changes in the affected peripheral nerves, but, as a rule, they are transitory, and, in our opinion, so far as they are not accidental complications (such as pressure paralyses), they are to be put in the same category with tabetic oculomotor paralyses (*vide infra*).

We may add, finally, that slight symptoms of motor irritation, slight twitchings in the muscles in the legs or in the fingers, are not uncommon, but they are noticed only when the attention is especially directed to them. It is not certainly known how they arise; perhaps they are of reflex origin, or perhaps they are connected with the ataxia and loss of muscle sense.

2. DISTURBANCES OF THE REFLEXES.—The involvement of numerous centripetal tracts in tabes causes, as we can readily understand, a number of very characteristic reflex disturbances. The remarkable peculiarity is shown here that the deep reflexes are interfered with much earlier and more regularly than the cutaneous reflexes. The cutaneous reflexes show no constant changes in tabes. They are usually approximately normal, but sometimes they are diminished, especially if there be at the same time a marked disturbance of sensibility. In many respects their condition corresponds to the condition of the sensibility to pain. The abdominal and cremaster reflexes are often preserved for a very long time.

The absence of the tendon reflexes, especially of the patellar and Achilles-tendon reflexes, is, however, an almost constant sign of tabes, and is a sign of the highest diagnostic value. As we have already said, the disappearance of the patellar reflex is one of the earliest symptoms of the disease, and is therefore of the greatest significance in the diagnosis of initial tabes. Since this symptom (primarily only the patellar reflex) was first discovered by Westphal it is not infrequently called "Westphal's sign." There are certainly cases of tabes in which the patellar reflex may persist for a long time, in spite of the development of many other morbid symptoms. These, however, are only very rare exceptions, which do not disprove the rule, and do not at all contradict our general views as to tabes. In individual cases those fibers which serve to set free the reflex may be spared for a long time, just as any other characteristic symptom of the disease may under some circumstances occasionally be absent. It is very important in every case to examine not only the patellar reflex, but also the Achilles reflex. We can confirm the observations of others that the Achilles reflex often disappears even earlier than the patellar reflex. Probably the loss of the reflexes is not infrequently preceded by a stage of increase. This is the reason why at the very onset of tabes we may find increased tendon reflexes associated with reflex immobility of the pupil, or occasionally an active patellar reflex with loss of the Achilles reflex. Concerning the precise anatomical cause of the disappearance of the patellar reflex, it can be due only to a degeneration in the centripetal portion of the affected reflex arc—that is, only in the fibers which belong to the territory of the posterior lumbar and sacral roots. As we have previously mentioned, the fibers for the patellar reflex enter the spinal cord about the level of the second to the fourth lumbar roots, and those for the Achilles reflex about the first and second sacral roots. The tendon reflexes of the upper extremities are frequently but not constantly absent. The direct mechanical excitability of the muscles, naturally, is not impaired in tabes, since it depends on a stimulation of the intramuscular motor nerves and the muscles themselves.

3. Condition of the Muscular Tonus.—A third result of the loss of the normal centripetal stimuli, which may be observed in the muscles, is the loss of tonus (hypotonia). It can be noticed in the characteristic response of the muscles to passive motion. In most cases we observe a very striking flaccidity of the limbs, so that scarcely any muscular resistance is felt. In tabetics who are confined to bed we can, for example, frequently flex the extended legs with ease at the hip to such a degree that they touch the head. Furthermore, the legs can be readily abducted very far at the hip, the knees can be bent so far with the patient lying on his abdomen that the heels can be made to touch the buttocks, and plantar and dorsal movements of the feet can be performed to an unusual degree and with great ease. We are dealing evidently, therefore, with a diminution of the muscular tonus—a so-called hypotonia of the muscles. Since all circumstances point to a reflex origin of the muscular tonus, the loss of the latter in tabes must be explained by a loss of those centripetal stimuli which exert a continuous tonic stimulation on the motor ganglion cells. It is to be noted that the condition of the muscular tonus does not always go parallel with the condition of the tendon reflexes. We have repeatedly observed a distinct loss of muscular tonus (hypotonia) in cases in which the tendon reflexes were still preserved or even very active (*vide supra*).

4. Disturbances of the Cutaneous and Muscular Sensibility.—While we have become acquainted so far only with those symptoms which are produced in the motor domain as a result of the loss of centripetal stimuli which, under normal conditions, are continually passing into the motor cells, we will now turn to the direct disturbances of conscious sensibility. As we have already said, tabes begins, in the great majority of cases, with symptoms of sensory irritation, which usually persist in the later course of the disease. Besides the simple paræsthesia—the feeling of tingling and numbness in the legs, and sometimes, too, a similar feeling which appears quite early in the upper extremities (especially often, as we have said, in the ulnar region)—the tabetic pains are remarkably characteristic of the disease.

The intensity of the pains differs very much in different cases; but we very rarely see a complete absence of them. The patient's attention is often first called to his slight and infrequent pains by direct questioning; but in some cases the severe pains are a constant distress to him. The pains most characteristic of tabes are the lightning-like, "lancinating" pains, which shoot like neuralgic pains for some distance along the course of the nerves. They often come on in very severe paroxysms ("pain crises," sometimes associated with considerable disturbance of the general condition, loss of appetite, etc.), last one or more days, and then disappear for a shorter or longer time. There are also boring, stabbing pains, which are fixed at one point and have their seat especially in the vicinity of the joints; and, finally, "constricting pains," which are felt most frequently in the back and loins. The well-known "girdle feeling" of tabid patients—that is, the sensation of a band tightly encircling the trunk, or a tight, "drawn-together" pressure on the lateral portions of the trunk—is a similar symptom of sensory irritation. The girdle feeling is manifestly due to irritative processes in the region of the lower dorsal, or upper lumbar, nerves. Since it is comparatively frequent, and often appears quite early, it also has a certain diagnostic significance.

The tabetic pains almost always show themselves first in the legs, but paræsthesia also occurs, and often very early, in the intercostal region (girdle sensation). In the ulnar region quite analogous pains sometimes appear, and in very advanced cases we have also observed pains in the region of the occipital nerves and of the trigeminus. On the other hand, neuralgic pains in the face, especially in the region of the frontal nerve, or in the occiput, or even migraine-like attacks, also occur, even in the initial stage of tabes, as we have ourselves observed. In very rare cases the lancinating pains in tabes may be accompanied by the appearance of herpes zoster.

Usually much later than the pains appears also a diminution of sensibility which can be made out objectively. As a rule, it may be stated that in most, but not in all, cases of tabes the sensibility does not remain normal, although well-marked anæsthesia never appears until the more advanced stages of the disease.

The form of the disturbances of sensibility varies extremely, and no disease furnishes so many opportunities for the study of interesting details in the region of anomalies of sensation as does tabes. Our knowledge of the occurrence of partial disturbances of sensation especially is very largely based on the examination of tabid patients. In order to form a correct opinion upon the condition of the cutaneous sensibility, it is therefore absolutely necessary to test carefully all the different qualities of sensation. Beginning with the examination of simple sensibility to contact (tactile sense), we often find this very well preserved even in advanced cases. There is often, of course, a certain blunting of sensibility. A slight touch with a camel's-hair brush is often, even in the early stages, no longer felt, or felt only very indistinctly, on the lower legs and feet, or on certain zones on the trunk (*vide infra*). The disturbance of the sensibility to pain is more marked. A blunting of the sensibility to pain in the legs is one of the most constant and early symptoms of tabes, but, in order to determine it, it is absolutely necessary to test at first only by brief pricks. We then find, as a rule, that such pricks cannot be positively distinguished from a touch with the head of the pin or with the finger, because the specific sensation of pain from the pin prick is lost. The case is quite different if we keep up a continuous pin prick.

In that case the patient at first usually feels only a painless touch, but after a short time (sometimes only some seconds later) there ensues often considerable pain accompanied by a reflex twitch in the leg pricked. This symptom was formerly explained by Naunyn, E. Remak, and others as "double sensation, due to "delayed conduction of the sensibility to pain," but, in our opinion, it is dependent merely upon the slow summation of pain irritation which requires a longer time (*vide* page 151), but there is also perhaps a delay in conduction.[1] Only in far-advanced cases can the analgesia, especially in certain parts of the skin, reach so great a degree that even continuous painful irritants can no longer excite a sensation of pain. The patient may be nearly or wholly insensible to deep pin pricks or strong faradic currents. Furthermore, a marked hyperæsthesia to painful stimuli is occasionally observed. Especially on the trunk, such hyperæsthetic zones (*vide infra*) are occasionally observed very early. It appears to be very plausible that a beginning disease of the sensory fibers produces first hyperæsthesia, later hypæsthesia, and, with complete degeneration of the fibers, anæsthesia. We have seen an analogous condition in the preceding discussion on the tendon reflexes.

The after-sensations of tabid patients are another peculiar symptom. It may happen that a patient after a single prick may have five, six, or more painful after-sensations at varying intervals.

Disturbances of the temperature sense are also quite frequently found, especially as partial disturbances of sensation, when the sensibility is otherwise well preserved. We should note particularly the occurrence of partial anæsthesia of the sensibility to heat or cold. On the other hand, the sensibility to temperature may sometimes be very keen, while in other respects there is quite a high degree of anæsthesia.

In addition to the diminution of temperature perceptions, we must also look for abnormal hyperæsthesia. Thus, often at the very onset of the disease, large zones are frequently found, especially on the trunk, that are markedly hyperæsthetic to cold, so that contact with a small piece of ice produces an abnormally marked and disagreeable sensation of cold.

Anomalies of the muscular sense (*vide* page 148), which are probably regularly present in every marked case of tabes, are of particular interest. The direction and amplitude of passive movements executed on these patients with their eyes shut, are recognized much more poorly and with greater uncertainty than in healthy individuals. In order to demonstrate this disturbance even in incipient cases, it is absolutely necessary to test the motions in all joints separately, and particularly not to omit the examination of the smaller peripheral joints (feet, toes). In the latter joints fairly marked disturbances can often be demonstrated, at a time when the movements of the larger proximal joints (knee, hip) are still felt normally. In advanced cases the loss of sensibility in the muscles and joints finally progresses to such a degree that the patient is absolutely undecided as to the position of his legs without ocular control. The patients do not notice if we place their legs in the most uncomfortable and unnatural positions. In the later stages of the disease the corresponding sensory disturbances also appear in the upper extremities. If the muscular sense in the arms be disturbed to a marked degree, the patient has considerable trouble, when the arms are previously stretched out, in bringing the hands together with his eyes shut. He gropes about in the air with his arms until he accidentally touches one arm with the other hand, and then he feels down this to the hand.

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[1] If, while the patient's eyes are shut, we prick the leg and the arm or neck as nearly as possible at the same time, the prick on the leg would necessarily be felt much later than that on the arm, if there is a delayed conduction of sensory impressions from the leg; but we have never as yet been able to make out this condition with certainty.

With the diminution of the sensation for passive motion, there is also generally associated a diminution of the "pressure sense." Deep pressure on the soft parts of the extremities, or on the periosteum and bone, is poorly perceived and not clearly differentiated from simple contact of the skin (*vide supra,* page 148).

In addition to examining the separate forms of sensibility, the determination of the limits of anæsthetic or hyperæsthetic zones is of interest, since it permits us to make deductions as to the extent of the degeneration in the different root zones. Attention has already been called to the fact that disturbances of sensibility generally affect the lower extremities first and the upper only later on. Recently, however, it has been particularly observed how frequently girdling hypæsthetic zones on the trunk can be demonstrated in incipient cases of tabes. Over the area of distribution of one or more dorsal or lumbar roots, on close examination we frequently find a marked diminution of the sensibility to touch, pain, etc. (Hitzig and Lähr and others). In isolated cases anæsthesia in the distribution of the occipital and trigeminal nerves may occur.

That the cause of all these disturbances of sensation is to be looked for in the disease of the peripheral sensory neurons needs no further argument. It is a question only what share the peripheral nerves and what share the spinal fibers and cells have in the causation, and how far in general such a localization of the symptom is justified. In general we regard it as most probable that the tabetic pains and paræsthesiæ are due to irritation in the processes of the spinal ganglia leading to the periphery—that is, in the sensory peripheral nerves. Anæsthesia must, of course, arise from any degenerative destruction of sensory fibers. We must also mention the fact that the peripheral nerve trunks, especially the ulnar nerve at the elbow, are often insensitive to direct pressure (ulnar phenomenon [Biernacki's symptom]).

5. DISTURBANCES IN THE EYE AND THE OTHER ORGANS OF SPECIAL SENSE.—The justification for regarding tabes as a combined systemic disease lies in the frequency with which certain cerebral symptoms, as well as the spinal, are found in it.

The symptoms in the eyes deserve the first attention. We find, of course not in all cases, but still in the great majority of them, that the pupils show no trace of contraction to light, although the well-known changes in the pupils are, as a rule, yet not invariably, very manifest upon any variation of the accommodation—dilatation of the pupils on looking at distant objects with approximately parallel axes of vision and contraction of the pupils on fixing a near object with the greatest possible convergence of the eyeballs. We give this phenomenon, first described by Argyll Robertson, but the precise anatomical cause of which is not yet known, the name of reflex immobility of the pupils with retained mobility on accommodation [reflex iridoplegia].

Here again we are evidently dealing with a reflex disturbance due to a destruction of centripetal fibers, which is consequently entirely analogous to the many other sensori-motor symptoms of tabes. It is a very characteristic feature that the immobile pupils are often not perfectly round, but on careful inspection seem rather irregular or polygonal. As we have already said, the immobility of the pupils is often a very early symptom, so that it also has a very great diagnostic importance, the more so that it is rarely found except in genuine tabes and the allied general paralysis. In regard to the average size of the pupils, they are often about normal, but we very often find both pupils unusually contracted (spinal myosis) or more rarely dilated. Quite frequently we find in tabes inequality of the pupils and, as a rare symptom, a striking change in the size of the pupils, so that first one and then the other pupil is the larger ("springing pupil"). We may also add that the dilatation of the pupil, which is normally produced by painful irritation of the skin of the cheek, is often absent in tabes (Erb).

Although much less frequently, it is also in the region of the eye that true motor disturbances—i. e., true paralysis of the external ocular muscles (see Fig. 163)—occur. They are usually unilateral, but sometimes bilateral, and often appear even at the beginning of the disease, so that diplopia may be the first subjective symptom of which the patient complains. In every sudden oculomotor or abducens paralysis, coming on without any other cause, we must think of the possibility of an incipient tabes. It is remarkable that these paralyses in many cases disappear permanently and entirely after some time, but it is always possible that they may recur later. We once saw a case of tabes with pronounced periodical oculomotor paralysis (*vide* page 229). The oculomotor paralysis, however, when it has once appeared, may be permanent, as we have repeatedly seen, especially in a case with bilateral abducens and unilateral oculomotor paralysis, and also in a case with almost complete bilateral oculomotor paralysis. In the autopsies on such cases we find the trunks of the affected nerves and their nuclei markedly atrophic, but it is very probable that the temporary ocular paralyses in tabetic patients depend upon changes in the peripheral nerves of the ocular muscles.

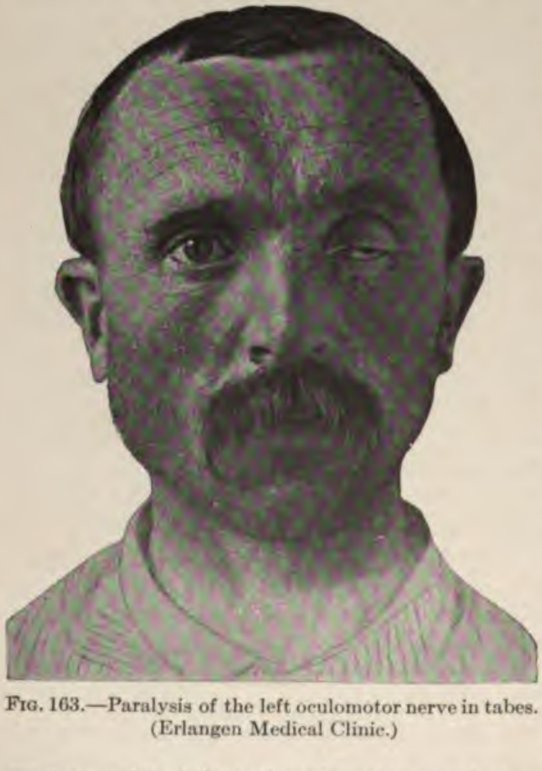

FIG. 163.—Paralysis of the left oculomotor nerve in tabes. (Erlangen Medical Clinic.)

The third complication in the eyes in tabes is optic atrophy. It occurs in about ten or fifteen per cent of all cases, and is usually an initial symptom, at a time when the absence of the tendon reflexes, which may usually also be noticed, is the only thing, except this, to render the diagnosis of the disease possible. The patient complains of diminution of vision; and the power to distinguish colors, especially green, disappears quite early. On objective examination, we find, besides this anomaly in the color sense, usually a limitation of the field of vision, and the beginning atrophy of the optic nerve can easily be made out on ophthalmoscopic examination. The affection sometimes makes little halts and slight apparent improvements, but it usually ends with complete blindness. It is very noticeable that in those cases of tabes which begin with an optic atrophy the other tabetic symptoms (pains, ataxia, etc.) often develop remarkably late, and only to a comparatively mild degree. The optic atrophy more rarely may not appear until the later stages of the disease, when all the other symptoms are already fully developed.

Auditory disturbances are much rarer than those of sight, but they also occur. The cause, in at least a part of the cases, is an atrophy of the acoustic nerve. We had a patient with tabes under treatment for a long time who was finally entirely blind in both eyes, entirely deaf in both ears, and almost entirely anæsthetic up to the forehead. The only possible way of communication was by writing the different letters of each word on his forehead with the finger! Symptoms are also frequently seen which resemble those of Ménière's disease—tinnitus, vertigo, and deafness.

Changes in the senses of taste and smell have been observed only in a few cases.

6. DISTURBANCES IN THE BLADDER, THE RECTUM, AND THE SEXUAL ORGANS.—Difficulty in emptying the bladder is an almost constant symptom in the later stages of tabes. It sometimes, indeed, appears very early. The nature of these disturbances still requires further study. In all probability the vesical disturbances of tabetics are due to the impairment of centripetal (sensory, reflex-exciting) nerve fibers of the bladder. The patients are unable to estimate with certainty the degree of distention of the bladder; they void their urine more infrequently and less completely than normally. Since the reflex mechanism for emptying the bladder is disturbed, the patients must strain with the abdominal muscles, and the bladder cannot be completely emptied. If the reflex tonus of the sphincter become weaker, dribbling of the urine and dribbling after voiding ensue. In some cases symptoms of severe sensory irritation of the bladder (so-called vesical crises) occur. A cystitis very often develops finally in conjunction with all these disturbances, which may be the starting-point of severe pyelocystitis and pyelonephritis, and thus be the cause of death.

Persistent constipation is also a very frequent symptom of tabes, the reason for which is perhaps to be sought in the defective reflex excitation of intestinal peristalsis. The constipation may in many cases give rise to very painful sensations in the loins and abdomen. The coccygodynia, which sometimes occurs in tabes, has already been mentioned (see page 183). Incontinence of fæces occurs quite rarely in the last stages of the disease.

A diminution in sexual power is found almost constantly in advanced cases, and it is also often one of the initial symptoms.

7. Symptoms in the Internal Organs.—We see, not very infrequently, in tabes certain nervous symptoms in the internal organs which are in part very characteristic. The most important and the most frequent are the so-called "gastric crises." These almost always come on suddenly and paroxysmally, and consist of an extremely severe cardialgic pain, which is accompanied by violent vomiting. The vomitus usually consists of fluid slimy masses stained with bile, and usually, though by no means invariably, contains so much hydrochloric acid that we can call it an actual hypersecretion (*vide* Vol. I, page 558). The vomitus contains blood in isolated cases. During the gastric crisis patients feel very wretched; they are restless and excited, and look pale and exhausted. The pulse is generally accelerated, and occasionally distinctly irregular. There is complete anorexia with consequent rapid emaciation. In one case of unusually severe gastric crises, I observed albuminuria during every attack. The attacks last about two or three days, but, in severe cases, eight to ten days and even more. In many patients they are repeated every few months. As we have said, the gastric crises may appear very early. We ourselves know cases where, in consequence of severe gastric crises, a serious gastric affection, or cholelithiasis, has been falsely diagnosticated in the beginning of the disease. Attacks of nervous diarrhea, "intestinal crises," usually, though not always, associated with colicky pain, have also been repeatedly observed.

We term attacks of severe dyspnœa "laryngeal crises." These depend perhaps upon a (reflex?) spasm of the glottis, and may attain a very alarming degree. They are also associated with a severe spasmodic nervous cough like an attack of whooping-cough. These laryngeal crises may come on so suddenly ("ictus laryngis") that the patient may sink to the floor with symptoms of the most extreme suffocation. Attacks of swallowing spasm have been described as "pharyngeal crises." We may also designate as pharyngeal and œsophageal crises particularly severe painful sensations which radiate from the epigastrium to the region of the pharynx. Paralysis of the laryngeal muscles (posterior crico-arytænoids) has also been observed, and it is analogous to the other tabetic paralyses (ocular muscles, etc.). We may assume changes in the vagus-accessory nucleus, or in the vagus or recurrent itself (Oppenheim), as the anatomical cause of all these symptoms.

In a few cases "renal crises" ("*crises néphritiques*") have also been described. They consist of severe attacks of pain, like renal colic. French authors also describe "urethral crises" and "*crises clitoridiennes*," the paroxysmal appearance of voluptuous feelings with a vaginal secretion in women, in the beginning of the disease.

We may mention, finally, that we sometimes see in tabetic patients a constant and very great frequency of the pulse, 100 to 120 a minute. This phenomenon may sometimes be purely nervous in origin; but it is generally due to coincident organic changes in the circulatory apparatus. It has been noticed for a long time how frequently the symptoms of aortic insufficiency, aortic sclerosis, or aortic aneurism, are found associated with tabes. Within the last few years, in which the diagnosis of incipient or rudimentary tabes, as well as the diagnosis of disease of the aorta, has become more exact, the frequency of this association has become more apparent. In some cases the symptoms of tabes may dominate the clinical picture; in others, those of the cardiac lesion, and only an accurate physical examination will reveal the presence of the other symptom-complex. An explanation for this remarkable combination of two so apparently dissimilar diseases can be found only in the assumption of a common ætiology—that is, in a preceding syphilitic infection. Tabes is frequently combined with syphilitic aortic sclerosis and its sequelæ (aortic insufficiency, aneurism). Conversely, as we have proved in cases of aortic sclerosis, aortic aneurism, and aortic insufficiency, if we search, we will very often find isolated tabetic symptoms, such as loss of pupillary reflex, absence of Achilles or patellar reflex, lancinating pains, hypotonia of the muscles, etc. This combination is therefore an additional proof of the doctrine of the syphilitic origin of tabes.

8. Trophic Disturbances.—Although trophic disturbances are wholly absent in many cases of tabes, they may, on the other hand, occur so frequently and in such manifold forms as actually to be classed among the most interesting, although least understood, symptoms of tabes. We must first mention that the general nutrition of many patients with tabes, although of course not of all, gradually suffers in a marked fashion. The patients grow thin and have a peculiar suffering, faded look. We cannot wholly deny a possible connection between this symptom and the primary disease. The trophic disturbances in the different tissues are still more striking. We have already spoken of the occasional appearance of herpes zoster. In some cases a marked exfoliation of the epidermis has been seen, and also a falling out of the hair, nails, and teeth. Peculiar paræsthesiæ and hypæsthesia of the mucous membrane of the jaws and gums often precede the loosening and falling out of the teeth. Sometimes there are small hemorrhages, apparently spontaneous, into the skin or into the visible mucous membranes, especially into the conjunctiva, as we have seen in several cases. The so-called *mal perforant du pied* is the worst trophic disturbance of the skin; this is a round, deep ulceration, situated on the ball of the foot or on the heel, which is very difficult and slow to cure in spite of the most careful treatment.

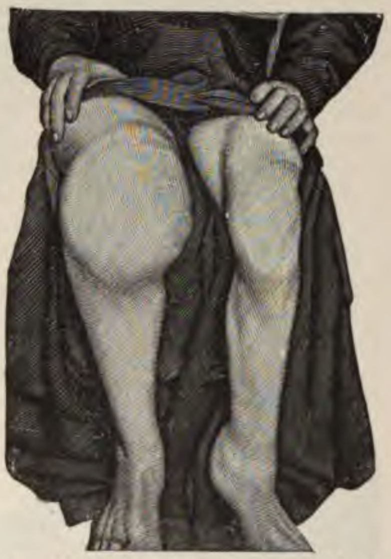

Fig. 164.—Tabetic arthropathy of the right knee and left ankle. (Personal observation.)

The most interesting trophic disturbances in tabes, however, are sometimes found in the bones and joints. These are the tabetic osteopathies and arthropathies, first accurately described by Charcot (Fig. 164). In the bones there sometimes develops a peculiar brittleness, which may lead to a sudden fracture, which apparently occurs quite spontaneously or on the slightest occasion, even in bed. The painlessness of these fractures, which is due to the tabetic analgesia, is very striking; the bones get out of place without the patient's noticing it, and thus the fracture often heals with displacement and an unusual callus formation. The tabetic joint affections are found most frequently in the knee and hip, more rarely in the foot, shoulder, or even in one thumb. They often seem to begin very acutely, a very great serous effusion sometimes taking place into the cavity of the joint. Besides this effusion there gradually develop the manifold processes of a chronic arthritis deformans—on the one hand, atrophy of the articular ends of the bones; on the other, thickening of the capsule, the formation of osteophytes, etc. In connection with the arthropathy there is often permanent dislocation of the joint. Thus we often see, following a tabetic affection of the knee, a *genu recurvatum* (Fig. 165). Arthropathic muscular atrophy is often seen, and is worthy of note (atrophy of the quadriceps in disease of the knee joint, etc.). Tabetic arthropathy may thus finally lead to monstrous changes in the joints such as can scarcely be found under other circumstances. Corresponding changes are also seen in the vertebral column. They may give rise to fracture of the vertebræ, spinal curvature, etc. The development of the so-called *"pied tabétique"* (Charcot) is also an osteopathic process; this consists chiefly of a considerable thickening and protrusion of the dorsum of the foot with a marked flattening of the sole.

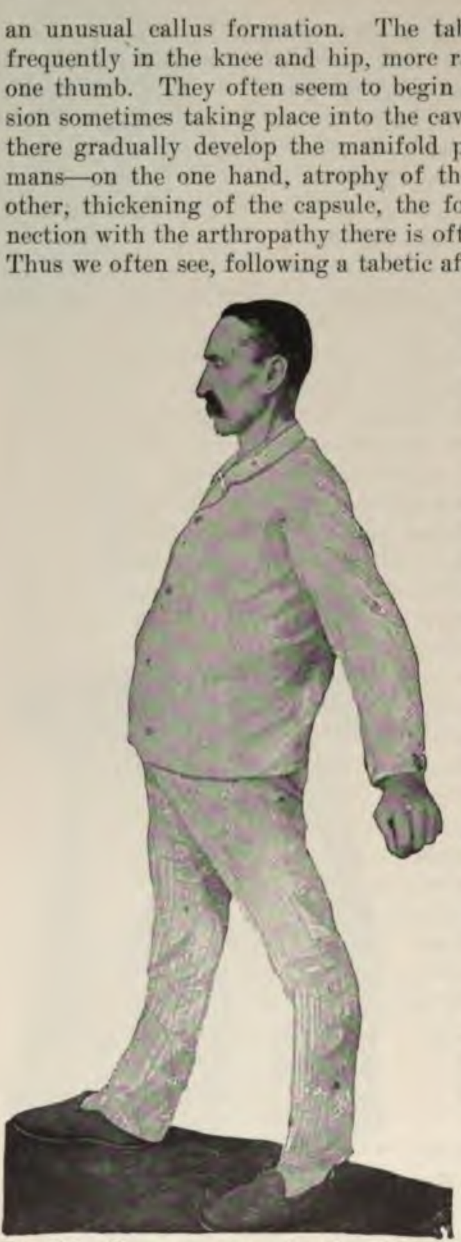

Fig. 165.—Genu recurvatum in tabes dorsalis. (Personal observation.)

We are not yet able to explain fully the occurrence of all these changes. It is hard to avoid entirely the assumption of trophic nervous disturbances. It has been claimed that degenerative changes have been found in cases of tabetic osteopathy and arthropathy in the nerves leading to the bones and joints; but it is also possible, on the other hand, that many of the so-called tabetic arthropathies may be of syphilitic origin. It is of the greatest importance, at any rate, that conditions may exist in tabes which must have a very unfavorable influence upon any existing bone or joint affection. The most important of these is analgesia. That is the reason why patients, in spite of a beginning arthropathy, do not spare the affected joint, but continue to irritate it still more. We saw a patient, for example, where an affection of the knee joint developed in a comparatively very early stage of tabes, which up to that time had not been diagnosticated at all. As the patient felt no pain whatever in his knee, he hunted most vigorously through a whole autumn, until finally an extremely severe swelling of the knee joint and an actual subluxation of the leg ensued. It is also possible that pronounced ataxic movements may frequently aid in irritating the articular surfaces.

The muscles preserve in general their normal state of nutrition, except as they take part in some joint affection or in general emaciation. Charcot described a case of a combination of tabes with genuine progressive muscular atrophy, in which the autopsy showed a degeneration of the anterior gray cornua in the spinal cord besides the atrophy of the posterior columns. The first report upon a unilateral atrophy of the tongue, which sometimes develops quite early in tabes, is due to the same observer. These and similar atrophies sometimes seen in other muscular territories are due to degeneration of the corresponding motor nuclei (hypoglossal nucleus, etc.); but peripheral neuritis may also lead to muscular atrophy in tabes. Joffroy described a special form of tabetic clubfoot (*"pied bot tabétique"*), which seems to be due to an atrophy of the calf muscles.

Some observations of apparently spontaneous rupture of the tendons (Achilles tendon, ligamentum patellæ) show that peculiar trophic disturbances may also develop in them.

9. CEREBRAL SYMPTOMS.—Besides the frequent important disturbances on the part of certain cerebral nerves, such as the optic and oculomotor, which have already been discussed, we must mention here the relation between tabes and progressive general paralysis (*q. v.*). On the one hand, the symptoms of tabes are often present in the course of general paralysis, so that the autopsy shows a typical degeneration of the posterior columns (Westphal); and, on the other hand, it also happens that the whole process begins with tabes, which may exist alone for years without any mental symptoms, and then only at the close do the symptoms of paralytic dementia (mental weakness, disturbances of speech, paralytic attacks, delusions of grandeur, etc.) appear. This combination of the two diseases is nothing strange, since general paralysis is also a metasyphilitic nervous disease, and is to be regarded as almost wholly analogous to tabes. Paralytic dementia has been called, not without reason, "tabes of the brain."

The complication of tabes with hemiplegia repeatedly occurs. The latter depends upon a cerebral hemorrhage or an embolic or thrombotic softening, so that the two diseases are probably connected only so far as they are both perhaps related to previous syphilis. It is worthy of note that in two such cases we saw scarcely any contracture develop in the paralyzed limbs.

10. CHANGES IN THE CEREBRO-SPINAL FLUID.—In the cerebro-spinal fluid obtained by Quincke's lumbar puncture, we have an index of the condition of the central nervous system, which is entirely similar, though not quite as reliable as that furnished by the urine as to the condition of the kidney. The most constant and important change of the spinal fluid is its comparative richness in lymphocytes (Widal, Schönborn, and many others), which can generally be demonstrated directly by a microscopical examination of the sediment after it has been collected in the centrifugal apparatus. It is to be noted that the same lymphocytosis occurs (though not in so marked a degree or as regularly) in secondary and tertiary syphilis. In the healthy, in purely functional neuroses and in noninflammatory diseases of the central nervous

system (for example, multiple sclerosis), an increased lymphocytosis is only exceptionally found. It is of great diagnostic importance that the lymphocytosis of the spinal fluid is rarely absent even in initial and rudimentary cases of tabes. Nonne and Apelt have demonstrated another chemical change. If we mix equal parts of spinal fluid and a hot saturated, filtered, and then cooled solution of ammonium sulphate, after about three minutes we obtain in most cases (according to Nonne in ninety-six per cent) a distinct cloudiness due to a precipitation of proteid substances (globulins?). In healthy people and in functional neuroses the reaction is absent; in other nervous diseases the reaction occurs not infrequently, but still with considerably less frequency than in tabes and paresis.

Course and Prognosis.—Although most of the characteristic symptoms of tabes appear in almost all fully developed cases, still, the order and the intensity of their onset vary greatly. We have stated that the initial period is usually characterized—apart from the symptoms that can be made out only objectively, such as absence of the patellar reflex and reflex immobility of the pupils—by the lancinating pains; that these may differ very much in intensity, and that the duration of this first stage may vary between a few months and ten or twenty years. The optic atrophy, the ocular paralyses, gastric crises, vesical disturbances, etc., were mentioned as rarer initial symptoms. The passage from the first stage to the second—the stage of ataxia—is sometimes very gradual, but in other cases very rapid and sudden. We have repeatedly seen such transitions. Frequently they come on clearly after some special exciting cause, as after some injury, some physical overexertion, some acute disease, etc. If the previous symptoms were slight, the patient dates his disease from this point, and says that he was quite suddenly broken down by some cause, and that since then he has not been able to walk at all, or else only with difficulty. Even in such cases there is often slow improvement following the sudden change for the worse, but the gain, of course, is not permanent. In other cases, however, the ataxia develops only very slowly and never reaches a very marked degree. Experience teaches, in particular, that tabid patients with early atrophy of the optic nerves or with unusually severe attacks of pain are often without more than a trace of ataxia for a very long time—many years!

No rules of general value can be given as to the further advance of the disease—the invasion of the arms by the ataxia, or the occurrence of the rarer symptoms, such as the joint affections, etc. Almost every individual case affords its idiosyncrasies, since one group of symptoms is often especially prominent, while another is entirely absent or developed only to a slight degree. On the whole, however, we can almost always make out a gradual, even if a very slow, advance in the disease. New symptoms appear, the old ones increase, the general condition becomes worse, until finally the last stage of the disease comes on.

Recovery from tabes in the sense that all the existing symptoms (immobile pupils, absence of patellar reflex, etc.) wholly disappear, have never yet been observed with certainty, and *a priori* such a thing is hardly conceivable; but it is not so very rare for the tabetic process to be arrested for at least a year. We have become acquainted with such cases only since we have been able to recognize with certainty the initial stages of the disease. We have ourselves known for many years a number of mild cases of tabes in whom it is scarcely possible to detect any recognizable progress in the disease and who regard themselves as almost perfectly well. They go to their business and have little or no trouble from their disease. Rudimentary cases of tabes also occur, in which the metasyphilitic intoxication produces only immobility of the pupil, loss of tendon reflexes, etc., and then to all appearances ceases to have any further action. With the advance in the diagnosis of tabes we have learned to recognize the frequency of these rudimentary cases. Thus, for example, I have previously mentioned how frequently other metasyphilitic diseases (aortic sclerosis, etc.) can be shown to be associated with isolated symptoms of tabes (loss of the pupil reflex, absence of tendon reflexes, lancinating pains, etc.). I scarcely believe that I am mistaken in asserting that the grand total of these rudimentary tabetics is far greater than the number of fully developed cases with severe tabetic symptoms. Of course we must be prepared for an ultimate change for the worse even in such cases.

In more advanced tabes, treatment may at most cause some improvement, delay the course of the disease, and alleviate certain symptoms; many patients, especially under favorable external conditions, may even in this stage lead a tolerable existence for years, but any marked improvement in their conditions can no longer be expected; on the contrary, there is generally an unmistakable gradual increase in the symptoms.

Diagnosis.—There is scarcely any other disease of the spinal cord whose diagnosis can in most cases be made with so great certainty and such comparative ease. Since tabes is a combined systemic disease, it affords a definite combination of symptoms such as can occur under no other conditions. The diagnosis, therefore, is scarcely ever to be made from any one single symptom, but only from the combination of all and from the whole course of the disease.

The diagnosis of initial or rudimentary tabes is especially important. In every case of obstinate "rheumatic" pains, or similar pains in the lower extremities, we should think of the possibility of tabes, and examine the tendon reflexes and the pupils. The combination of characteristic pains, absence of the patellar reflex on the two sides, or absence of the Achilles reflex, and reflex immobility of the pupils, usually makes the diagnosis almost absolutely certain, as was said above; two of these symptoms, especially if the reflex immobility of the pupils be one, make it at least very probable. Ocular paralyses, temporary ptosis, or temporary diplopia, may be very important for the diagnosis. We should also remember that the disease may begin with optic atrophy, and that early gastric crises may simulate a gastric affection, early disturbance in micturition a vesical trouble, or swelling of the joints a joint disease, until careful examination of the other symptoms explains the true nature of the disease. The essential point is to think at once in every such case of the possibility of tabes, and look for the distinctive objective symptoms. Among the latter we would call especial attention here, besides the symptoms generally recognized (absence of tendon reflexes, immobile pupils, slight vesical weakness), to the limited girdle-like disturbance of sensation (either hypæsthesia or hyperæsthesia to cold) on the trunk, since in doubtful cases this is often of decided significance in diagnosis. It has already been mentioned that the changes in the cerebro-spinal fluid occur in the beginning of tabes, and are generally found to be marked even in the rudimentary stages.

In the fully developed ataxic stage of tabes the diagnosis is almost always easy, and it often can be made at the first glance. The history, the characteristic ataxic gait, the swaying with the eyes shut, the absence of the reflexes, etc., exclude every doubt. The diagnosis may be more difficult if we happen to see the patient for the first time in the final stage, when actual paralysis has set in, when a complicating hemiplegia has arisen, etc. In such cases we must lay stress on the development of the disease and find out what characteristic tabetic symptoms—pupillary symptoms, absence of patellar reflex, vestiges of ataxia, or pains—can now be discovered. With proper attention and knowledge of the case the diagnosis can even then almost always be made correctly.

Vertebral affections are to be mentioned first of the diseases which may be confused with tabes. These also cause, under some circumstances, lancinating pains and an absence of the patellar reflex, as a result of compression of the spinal roots; but in these cases the later course of the disease is entirely different, apart from the changes in the vertebral column and the absence of other characteristic symptoms of tabes, especially the immobile pupils. The same holds true of certain deep-seated tumors in the vicinity of the spinal cord. We have already said that in rare cases multiple sclerosis (*q. v.*) may offer symptoms similar to those of tabes. In these cases the chief stress in regard to diagnosis is to be laid on the condition of the tendon reflexes, the abdominal reflexes, the pupils, the disturbances of sensation, the fundus of the eye, and the bladder. It is of greater practical importance that certain toxic nervous diseases may have a great similarity to tabes. Chronic alcoholic neuritis has already been spoken of in this connection—the so-called pseudo-tabes of alcoholic subjects. In these cases, however, the reflex immobility of the pupils and the disturbances of the bladder are usually absent, while atrophic paralysis may develop at the same time, which hardly ever happens in tabes. The ætiological factors (alcoholism) are also, of course, to be taken into account. It may be mentioned here that we have twice seen a group of nervous symptoms in men who have worked many years in tobacco factories which resembled tabes in so many points that we might term it "nicotin tabes." Since, however, similar cases have not been subsequently observed by us or by others, we do not wish to attach much importance to these observations. In adults postdiphtheritic ataxia may be mistaken for tabes. In such cases a consideration of the ætiology, and especially the condition of the pupils and of the vesical disturbances, will usually make the correct diagnosis easy. "Diabetic tabes" (page 105) can scarcely, with proper attention, be mistaken for true tabes. The differential diagnosis between tabes and Friedreich's hereditary ataxia will be described in the next chapter.

Finally, the question may be occasionally raised whether we are dealing with incipient abes or a purely functional disturbance (neurasthenic pains, etc.). In these cases a cytological or chemical examination of the cerebrospinal fluid (*vide supra*) may sometimes be of decisive value.

Treatment.—The first question which is usually raised at present in regard to the treatment of tabes is respecting the efficacy of a specific antisyphilitic treatment. In our opinion, the ability to answer this question correctly depends not merely on recognizing the fact of the connection between tabes and syphilis, but also on forming a clear conception of the nature of this connection. There can then be no doubt that in tabes there is not even a remote question of such efficacy of mercury or iodid as we see almost as a rule in true tertiary, gummatous syphilitic diseases. We very often see in tabes no benefit at all from an inunction cure or from the internal use of iodid of potassium, and in some cases the condition seems even to grow worse under such treatment. Such observations were for a long time cited as the chief evidence against the connection between the two diseases. If, however, we adopt the position which we have advanced that tabes is a post-syphilitic degenerative nervous disease, just as ataxia may sometimes be a postdiphtheritic disease, we can easily understand the inefficiency of a specific remedy, which acts only on syphilitic new formation of tissue. Nevertheless, we may not wholly exclude antisyphilitic methods of treatment from the therapy of tabes; for, in the first place, it is always possible (and it seems to us to be confirmed by many trials) that an antisyphilitic treatment (inunction, iodid of potassium) may influence in a way beneficial to the patient the conditions which cause tabes to advance. It has, furthermore, been fully attested by repeated observations that, besides the tabetic changes in the cord, there may also be at the same time actual syphilitic gummatous processes, which of course may contribute their part to the symptoms of the disease. That these morbid processes require thorough specific treatment is certain, and therefore we regard it as justifiable and even desirable to make a trial of inunction in beginning tabes, and under some circumstances even in advanced tabes, especially if any anomalies in the type of the disease suggest the possibility of coexisting gummatous changes. Very experienced observers, especially Erb, have gained the impression that a careful inunction treatment sometimes visibly benefits the patient. We cannot, of course, report much that is favorable from our own experience, but we occasionally employ specific treatment, especially a course of inunctions with mercurial ointment, or prescribe iodin preparations in cases that seem suitable. Every inunction treatment prescribed in tabes must be carefully watched, that no injury be done to the patient by this vigorous treatment. The observation of many experienced oculists must be particularly borne in mind that with a beginning optic atrophy, mercurial treatment is often decidedly harmful. In most advanced cases of tabes, especially where the patient's general condition is feeble, we must abstain wholly from mercurial treatment.

If the antisyphilitic treatment be not indicated, or if it has been unsuccessful, electricity and balneotherapeutics or hydrotherapeutics deserve relatively the most confidence, although, of course, we must beware of exciting too sanguine hopes.

The electrical treatment consists chiefly of the passage of the ascending constant current through the spinal cord. The current must not be too strong, and the sittings should take place daily or every other day. Erb recommends placing the medium-sized cathode in the vicinity of the sympathetic and the large anode close to the spinous processes on the other side of the vertebral column, moving it at intervals from above downward. This procedure lasts about four or five minutes for each side. We also obtain good results symptomatically when there are severe pains, vesical weakness, etc., by peripheral galvanization. If we find painful points on the vertebral column, as is rarely the case, they should be especially treated with the stabile anode. The treatment recommended by Rumpf with the faradic brush has also been used at times with good results. This consists in brushing the skin of the back and the extremities with a strong current for five or ten minutes. Every form of electrical treatment, in order to obtain results, must be kept up for months.

Hydrotherapeutics, used rationally, have often resulted in considerable improvement in tabes, although they may cause much mischief. Hot baths, especially vapor baths, are often followed by a rapid change for the worse—a fact which, unfortunately, we can often observe where vapor baths have been prescribed for patients at the beginning of their disease "for rheumatism." Continuous wet packs and vigorous rubbings are also often accompanied by unfavorable results. Tepid half or full baths, however, of about five or ten minutes' duration, and from 75° to 86° F. at most (20° to 24° R.), associated with gentle rubbing of the skin, or brief cold sponging, etc., often do good service. Wet compresses, applied around the abdomen or the legs at night, often have a favorable influence on the pains. In general, it is a good plan to send well-to-do patients in summer to a sanitarium which is conducted by an experienced director and well equipped, but the necessary procedures may also be undertaken at home.

Of the baths whose use is recommended in tabes, Oeynhausen has the greatest reputation. The baths in Nauheim have a composition and action very similar to those at Oeynhausen. We have also seen very good results from the baths at Kissingen. Many physicians praise mud baths and iron baths (Pyrmont, Driburg, Cudowa, Elster, Franzensbad, etc.), while the indifferent thermal baths (Teplitz, Wildbad, Ragatz, etc.,), formerly much in favor, are at present rarely used in tabes. Concerning the artificial carbonic-dioxid brine baths, which are to be highly recommended in tabes, compare what has been said under the treatment of myelitis.

Besides the methods of treatment thus far mentioned, there are still a number of internal remedies, the use of which seems sometimes to be of advantage. The chief ones to be mentioned are nitrate of silver, first recommended by Wunderlich, $\frac{1}{6}$-gr. pills (gm. 0.01), at first three, gradually increasing to six a day, before meals; and ergotin,[1] 1-gr. pills (gm. 0.05), three to six a day; we may also try iodid of potassium, phosphorus, arsenic, etc. All these remedies, especially the two first named, may be used for a long time, and, with interruptions, even for years.

In consequence of a much too sanguine indorsement by Langenbuch, nerve stretching (usually the operative stretching of both sciatics) was for some time practiced on many tabid patients, but since experience has taught us that nerve stretching, in spite of some apparent successes, scarcely ever exerts a permanent favorable action, and is also not wholly without danger, the operation has been almost entirely given up in tabes. It may still be tried in those cases where we have unusually severe attacks of pain in the region of definite nerves. In such cases we should first try bloodless stretching (page 182). The "suspension treatment" of tabes has also had only a brief vogue. This consisted in hanging up the patient for a short time by an apparatus fastened under the chin [and arms], thus stretching the spinal cord and the nerve roots by the weight of the body. The successes at first reported are of very doubtful significance, while there were certainly some very unfortunate consequences. We ourselves cannot advise the use of this method. The treatment of tabes by firmly fitting corsets is also much praised and recommended. If we listen to the patients who have just left the sanitarium of some eminent orthopedic surgeon, they know no end to their praise. If we see them again in a year and a day, we find them ataxic as before, and the corsets—in some corner! Temporary symptomatic benefit should not be denied, but of course the corset treatment cannot affect the tabetic process itself.

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[1] There is only an apparent contradiction in the fact that, in spite of the occurrence of an "ergotin tabes" (*vide supra*), ergotin is also used as a remedy against tabes. It may very well be that the same remedy, which in large doses causes certain systems of fibers to atrophy, may in smaller doses have some favorable (stimulating) action on the same system of fibers; but we must always be cautious in the use of ergotin.

Symptomatically, the same remedies are to be considered as were mentioned in the treatment of chronic myelitis. We try to alleviate the pains by narcotic embrocations and bandaging the legs. Of internal remedies, aspirin and antipyrin, and also antifebrin, phenacetin, pyramidon, or a mixture of these remedies, sometimes undoubtedly alleviate and shorten the pains. [Chlorid of aluminium, in doses of 5 to 10 gr. (gm. 0.1 to 0.25) three times a day, occasionally relieves the pain.—K.]

Narcotic embrocations, a Priessnitz poultice, or a hot pack also occasionally afford relief to the painful limbs. We have several times used Bier's hyperæmic treatment for severe pains, apparently with good results. Some observers praise the action of the so-called *points de feu* (circumscribed burning of the skin with the thermocautery) along the spine at the points of exit of the affected nerve roots. In bad cases morphin is indispensable, but, considering the long duration of this disease, we ought to be very cautious in prescribing its use. We try to remove the constipation by prescriptions as to diet, or by mild cathartics, such as the bitter waters, tamarinds, and rhubarb, by enemata, or by electrical treatment and massage of the abdomen. Galvanic treatment often acts very favorably on the bladder symptoms. For these symptoms Charcot praises the action of ergot, and other physicians recommend strychnin. Morphin is the best remedy in the gastric crises. We may also try warm poultices, washing out the stomach, belladonna, chloral, lupulin, etc. The recent attempt to ameliorate severe gastric crises by section of the posterior dorsal roots is very interesting. Severe laryngeal crises also require morphin injections as a rule. Optic atrophy unfortunately defies any treatment. We usually try strychnin injections, or treatment with mild galvanic currents, to encourage the patient.

One method for the symptomatic treatment of tabes deserves special mention, and that is the treatment of ataxia by methodical exercises (H. G. Frenkel). If ataxic patients are systematically admonished to execute all the movements of the several joints slowly and as regularly as possible several times each day for a definite time, and also to perform certain other movements (touching a definite point with the fingers or toes, walking a chalk mark, walking between the rungs of a ladder, seizing swinging balls, putting stoppers into definite holes, etc.), we may often recognize that the practice actually has an influence upon the certainty of the movements and leads to an improvement in the ataxia. Certain sanitaria carry out this method of treatment with especial reference to the ataxic disturbances of each individual case and by the use of complicated apparatus. Occasionally the results of such a well-directed treatment are undoubtedly very good, but should not be overestimated.

It is also of the greatest importance that the physician should regulate and supervise the patient's mode of life in its general hygienic and dietetic aspect. We must warn him especially against any physical or mental overexertion, prescribe a prudent but strengthening diet, withdrawing all large amounts of alcoholic drinks, and enjoin good air, a country residence in summer, or a medium mountain climate, and in winter at times southern health resorts. How effective this general dietetic treatment of tabes is may be seen in hospitals, where tabetic patients of the lower classes are sometimes much benefited by simple rest and good care. The earlier we get the patient under treatment, the more persevering and careful should we be in our treatment, because then we can still hope for a certain success. In old and far-advanced cases we must confine ourselves to a purely symptomatic treatment.

CHAPTER VIII

HEREDITARY (JUVENILE) ATAXIA (FRIEDREICH'S DISEASE)

Ætiology and Pathological Anatomy.—In 1861 Friedreich first described a peculiar disease observed by him in several members of the same family. He at first regarded the disease as a special form of tabes on account of the marked ataxia attending it, but later investigations have shown that this "Friedreich's disease" has nothing in common with true tabes except a certain similarity of symptoms (especially ataxia), which is due to the fact that the anatomical changes in the cord agree in part in the two affections. While tabes represents an acquired degeneration of different neuron systems, Friedreich's disease is undoubtedly due to a congenital morbid predisposition of certain neuron systems, as a consequence of which these neuron systems have not developed their full and permanent functions and therefore atrophy slowly and prematurely. This congenital origin of the disease is most evident from the fact that the disease is not infrequently seen in several (two to five) brothers and sisters, and in some cases in members of two or more generations of the same family. Of course sporadic cases of Friedreich's disease are seen with comparative frequency, but in such cases the youthful age at which the first symptoms of the disease are noticed indicates the congenital abnormal tendency. The first symptoms sometimes appear at the age of seven to ten years, but frequently not until puberty, and in some cases even somewhat later (eighteen to twenty years). It is usually hard to determine accurately the period of onset, because the disease begins very gradually, and therefore its onset may certainly remain unnoticed for a long time. The disease is about equally common in the two sexes, perhaps a little commoner in the male. The first symptoms sometimes seem to follow some acute disease (scarlet fever, measles, influenza), injury, etc., but these are exciting causes which make the existing morbid tendency manifest.

Clinical History.—The first symptom invariably to be noticed, which is by far the most important later, is ataxia. It shows itself at first as a slowly increasing uncertainty of gait; almost at the same time or only a little later the ataxia is noticed in the upper extremities. When the disease is fully developed the gait is unusually characteristic. In regard to the ataxic movements of the legs the gait resembles that of tabes, but it usually differs from it in the much greater swaying of the trunk. In this respect the gait is more like that of the so-called cerebellar ataxia. The fact that in Friedreich's disease the muscles of the trunk and pelvis are also affected, and even to a greater degree, by the ataxic disturbance, is the reason why the patient usually manifests more of the so-called static ataxia than is the case in tabes—that is, that he continually sways very much when standing quietly (*vide* Fig. 166). The patient therefore always stands and walks with the legs wide apart to give the body a better support. If we make him stand still with the legs as close together as possible, we notice the constant muscular contractions necessary to maintain the equilibrium. With the removal of the visual control by closing the eyes, the uncertainty and swaying of the body increases. The ataxia is also very well marked in the legs, as is most readily shown by the well-known "knee-heel test," and also in the arms, as tested by bringing the finger tips together, by reaching for an object held before one, writing, etc. The disturbance of speech, which is often noticed in the later stages, is due to an ataxia of the muscles requisite for speech, and consists of an indistinct articulation and a slow, scanning, badly modulated manner of speaking. Nystagmus of the eyes, especially on looking far to one side, sometimes occurs as a further ataxic symptom, just as in multiple sclerosis, but it is by no means invariable, or at least it may not appear until late in the disease.

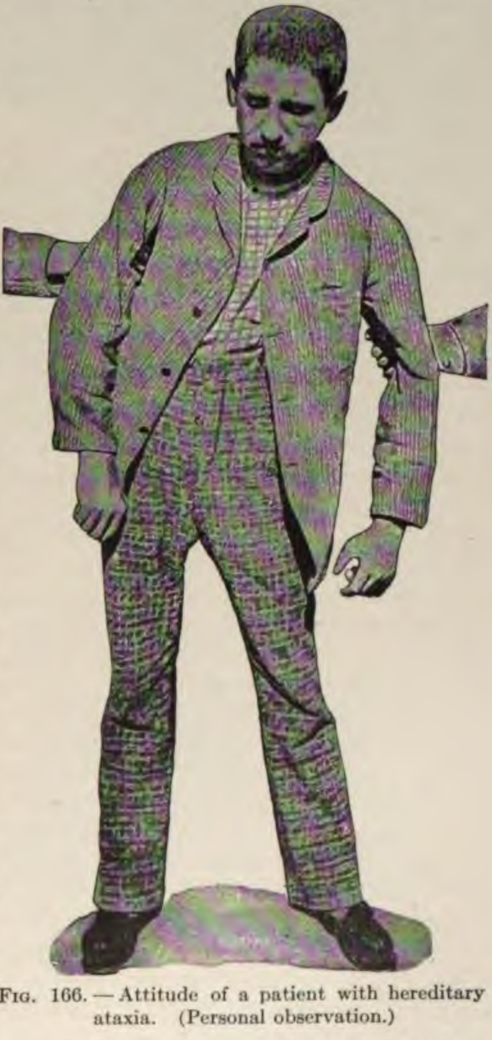

FIG. 166.—Attitude of a patient with hereditary ataxia. (Personal observation.)

Besides the ataxia, which finally becomes very pronounced, for a long period we can usually detect only one other important symptom, probably always of very early occurrence—complete absence of tendon reflexes (patellar and Achilles). All other nervous functions may remain wholly unaffected for a long time. The motor strength of the muscles is very well retained. We know a patient who, in spite of the most marked ataxia, has traveled several miles a day by the aid of a cane. The muscles are usually not remarkably well developed, but they show no striking atrophy, apart from rare complications. Many patients show a peculiar choreic muscular unrest. Athetoid movements of the fingers and peculiar shaking movements of the trunk have occasionally been observed. The sensibility of the skin and deeper parts (joints, muscular sense) may for a long time remain perfectly normal, a condition which has been brought forward, especially by Friedreich, as an argument against the dependence of ataxia upon sensory disturbances. Of course this preservation of sensibility must not be too much emphasized, since many other observers and we ourselves have repeatedly found slight disturbances of sensibility (especially on the feet, toes, and lower legs) on careful testing: for example, a diminution of the tactile sense, the pressure sense, particularly the muscular sense, etc. The sensibility to pain seems almost always to remain unaffected Paræsthesiæ and spontaneous pains, as well as the other varieties of tabetic crises, are also entirely absent. The cutaneous reflexes, aside from the Babinski [and Oppenheim] toe reflexes, which are occasionally present, show no material disturbance and the pupil reflexes are always preserved. The muscular tonus is, as a rule, not so markedly diminished as in true tabes. This absence of hypotonia and the presence of the Babinski [and Oppenheim] reflexes are often the only clinical signs which point to an involvement of the lateral tracts (*vide infra*). True spastic symptoms can never develop, since in all probability the involvement of the posterior tracts precedes the degeneration of the pyramidal tracts. As a rule, there are no marked vesical disturbances. In some cases, however, there may be atrophy of the optic nerves, just as in tabes. We may also note that the toes, especially the great toes, have a very striking tendency to assume a position of extreme dorsal extension, and we sometimes see a tendency for the foot to assume the equinus position. The vertebral column often shows an evident scoliosis.

The course of the disease is very chronic and it may last for years and years. The ataxia finally becomes so great that the patient is bedridden. In such cases there is finally actual paralysis, greater disturbance of sensation, not infrequently marked disturbances of intelligence, etc. Death often ensues from some intercurrent disease.

Pathological Anatomy.—Hereditary ataxia was the first form of disease in which a combined systemic lesion of the spinal cord was discovered by careful anatomical investigation by Kahler and Pick. Later observers (F. Schultze, Rütimeyer, and others) have confirmed these discoveries. We find the posterior roots and the posterior columns most diseased, and chiefly the zone of the entering root fibers. Lissauer's areas are usually exempt, in contrast to the degeneration of tabes. The cerebellar lateral tract (including probably the cells of Clarke's columns) is also regularly affected, and also Gowers' tract (see the chapter on Secondary Degenerations in the Cord) and the neighborhood of the pyramidal lateral tract, although in lesser degree (*vide* Figs.

167 *a, b,* and *c*). The histological process, as in all systemic diseases, consists of an atrophy of the nervous elements and a considerable secondary increase of the neuroglia.

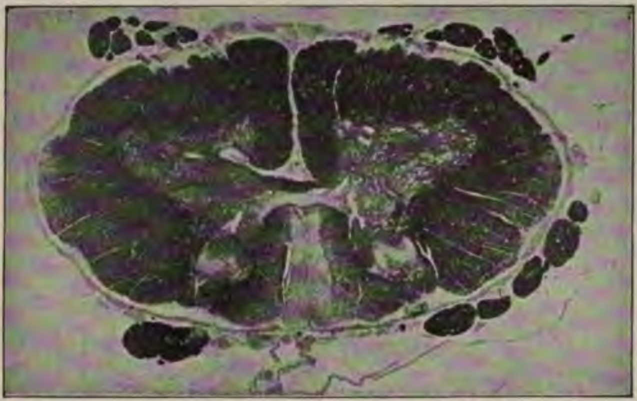

FIG. 167 *a*.—(Cervical cord.)

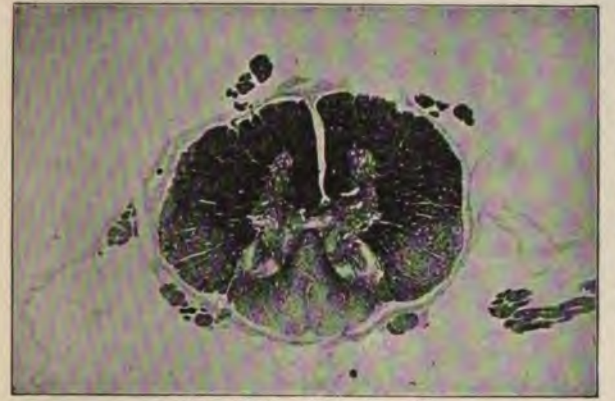

FIG. 167 *b*.—(Dorsal cord.)

It would be very interesting to make a careful comparison between the anatomical lesions and the symptoms in Friedreich's disease, on the one hand, and in tabes on the other; but we are still greatly in want of a sufficient number of careful observations. The ataxia of the extremities evidently depends on a degeneration of the posterior columns, and that of the trunk probably on a degeneration of the lateral cerebellar tracts. The lack of prominence of other lateral tract symptoms (with the exception of the Babinski [and Oppenheim] reflexes) has been already emphasized. The absence of lancinating pains in hereditary ataxia is striking. This is explained perhaps by the integrity of the peripheral nerves, but principally also by the different nature of the disease:

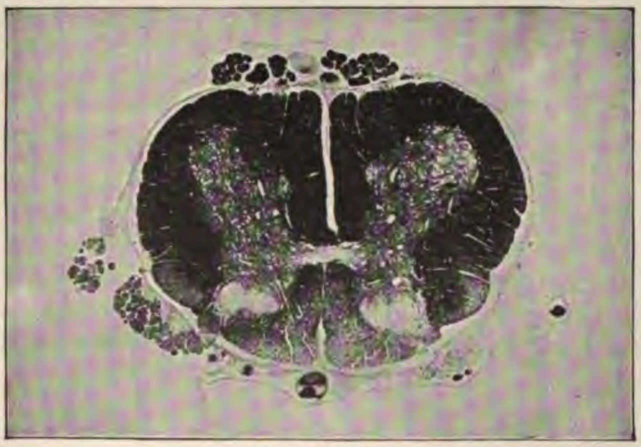

FIG. 167 *c*.—(Lumbar cord.)

Anatomical changes in the spinal cord in hereditary ataxia (Friedreich's disease). (Personal observation.)

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The disease has a very tedious course and usually lasts many years. The prognosis is absolutely unfavorable. Death ensues from some intercurrent disease or as a result of the ultimate general weakness. Anatomical examination, as has been said, shows as the most striking change an atrophy of the cerebellum, which is diminished about one half in size. The spinal cord is sometimes poorly developed on the whole, but it shows no columnar degeneration.

The disease is distinguished very essentially from Friedreich's hereditary ataxia, which otherwise is very similar to it, by this anatomical condition, which is, however, still in urgent need of thorough study. Clinically the two diseases are to be distinguished particularly by the condition of the tendon reflexes and the fact that cerebellar ataxia usually comes on somewhat later in life. The differential diagnosis between cerebellar ataxia and multiple sclerosis is often very difficult. The generic (family) occurrence is the chief distinction.

The treatment can be merely symptomatic, and it has little prospect of success.

CHAPTER IX

THE PRIMARY DEGENERATIONS OF THE MOTOR TRACT, INCLUDING THE MUSCLES

1. PRELIMINARY REMARKS

In the two preceding chapters we have become acquainted with two diseases whose anatomical basis is a primary degeneration of a number of systems of fibers whose conduction was chiefly *centripetal*. In tabes we found the cause of the degeneration to be exogenous, acquired noxious influences, coming from without and probably of chemical nature; in hereditary ataxia, on the contrary, the cause was endogenous anomalies—that is, anomalies in the embryonic predisposition, and therefore congenital—which caused a premature atrophy of nerve fibers which had from birth an abnormally weak power of resistance. In this chapter we shall become acquainted with a number of diseases, which are due chiefly to primary degeneration of the great *centrifugal* motor-conducting tract, especially the pyramidal tract. We will see that, just as with hereditary ataxia, the basis of the disease is chiefly an endogenous, congenital weakness of this portion of the nervous system, so that it is therefore very often a family, generic disease, although in some cases exogenous noxious influences are probably also to be considered.

In order to obtain a preliminary survey of the whole group of diseases here under consideration, we may picture to ourselves, as in the accompanying diagram (Fig. 168), the course of the long conducting tract leading from the central convolutions to the muscles as has been fully described on page 193. In the first place we will consider a form of disease in which ultimately the whole cortico-muscular conducting tract, from the cortical cell, *C*, up to and including the muscle, *M*, is found degenerated. This is *amyotrophic lateral* *sclerosis.* We will next describe a form of disease in which only the second portion (the so-called second neuron) of the whole tract—namely, the portion from the ganglion cell in the anterior horn of the cord (*V*) up to and including the muscle—is affected. This is *spinal muscular atrophy,* and the completely analogous *progressive bulbar paralysis.* In the third place we shall see that the first cerebro-spinal main portion of the tract, *PyS,* may be alone affected up to the cell of the anterior horn, but without involving it. We then have the true *primary pyramidal lateral sclerosis,* with the clinical picture of pure spastic spinal paralysis. Here the lateral pyramidal tract, in the stricter sense, may be exclusively involved (muscular hypertonia, increased tendon reflexes), or an involvement of the entire motor tract and the postero-lateral tracts may produce complete paralysis. In the fourth place the disease may involve only the peripheral motor nerve, *m,* with its corresponding muscles (*neurotic muscular atrophy*), and, finally, we may also place here by far the commonest form of disease, *progressive muscular dystrophy*—that is, that disease in which only the terminal apparatus of the whole tract, the muscle itself, undergoes a progressive atrophy.

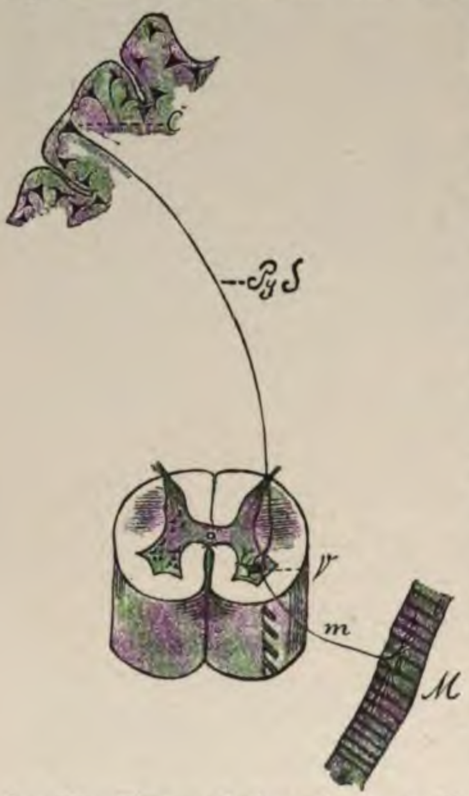

FIG. 168.—Diagram of the motor tract. *C.* Motor-ganglion cell in the cortex. *PyS.* Pyramidal lateral tract. *V.* Ganglion cell of the anterior horn. *m.* Motor nerve. *M.* Muscular fiber.

The forms of disease just mentioned are essentially, so far as they are endogenous diseases, all closely allied, but it is still necessary for a time to keep them separate, at least so far as they show very definite marks of distinction, both clinically and anatomically. Only with a more advanced knowledge of the special nature of all these morbid conditions can we make a strict ætiological division of them. At present we must remember that we cannot consider the different clinical and anatomical forms as different diseases, strictly separate from one another. The more our knowledge increases, the more prominent seem to be transitions and connecting links between the different forms.

2. AMYOTROPHIC LATERAL SCLEROSIS

Ætiology and Pathological Anatomy.—For the first accurate knowledge of amyotrophic lateral sclerosis we must thank Charcot, who published in

1869, in company with Joffroy, his first observations upon such cases, and in 1874 was able to give quite a complete description of the disease; but an exact knowledge of amyotrophic lateral sclerosis was first made possible by Flechsig's investigations upon the course of the conducting tracts in the spinal cord. From these we can now say that the disease is manifested anatomically essentially as a primary systemic degeneration of the whole corticomuscular conducting tract, and not only of the spinal portion thereof belonging to the extremities, but also of the bulbar portion belonging to the muscles of the face, tongue, soft palate, etc. These two parallel portions must be regarded as completely equivalent parts of one great motor system—a point to which we shall frequently return later. The motor-nerve nuclei in the medulla and pons (for the hypoglossus, facial, motor trigeminus, etc.), and also the nuclei for the oculomotor nerves, are to be regarded as precisely analogous to the ganglion-cell groups in the anterior horns of the cord for the muscles of the extremities.

In amyotrophic lateral sclerosis a large part of this motor-conducting tract undergoes degeneration, and sometimes, as it seems, throughout almost its whole extent. If we examine the nervous system in such a case we find in the spinal cord, especially in the cervical region, that the gray anterior columns are narrowed and sunken, that the large ganglion cells of the anterior horns in the cervical and lumbar cord, or in the cervical cord only, are atrophied and in part wholly absorbed (Fig. 169). We find the pyramidal tracts greatly degenerated in both lateral columns, and, of course, in one or both anterior columns when there is an anterior pyramidal tract; but in the other segments of the antero-lateral tracts there is generally a pronounced destruction of nerve fibers as well, which, however, is not to be wondered at, if we consider that numerous motor-fiber tracts also pass through this area. In contrast to the completely (or nearly) normal posterior columns and posterior horns, the entire anterior motor segment of the spinal cord appears flattened and diminished in size (*vide* Fig. 169). In the anterior horns also the number of medullated fibers, part of which are certainly processes of the pyramidal lateral tract (*PyS*) [1] fibers, is diminished. Farther upward, as was first discovered by Charcot and Marie, we can follow the degeneration of the pyramidal tract, not in all, but certainly in many cases, through the pyramids of the medulla, the pons, the crura, and the inter-

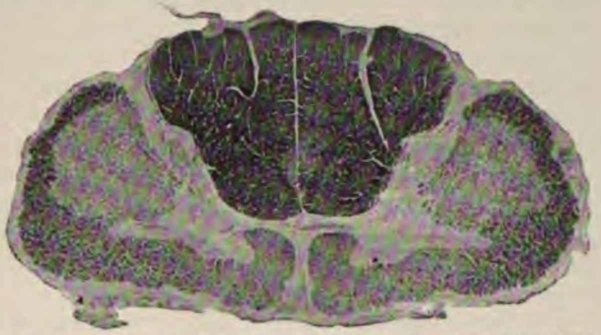

FIG. 169.—Section of the cervical cord in amyotrophic lateral sclerosis. Degeneration of the pyramidal tracts, in part of the other antero-lateral tracts, pronounced atrophy of the anterior horns. (Personal observation.)

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[1] Abbreviations in frequent use in what follows: PyS = pyramidal lateral tract (Pyramiden-Seitenstrangbahn); PyV = anterior pyramidal tract (Pyramiden-Vorderstrangbahn); KlS = lateral cerebellar tract (Kleinhirn-Seitenstrangbahn.)

nal capsule, to the central convolutions, in whose ganglion cells we can also find atrophy. The bulbar motor nuclei, especially the hypoglossal nucleus and the vagus-accessory nucleus, more rarely the facial nucleus, etc., are also atrophied, as a rule, just like the anterior horns of the cord. From all these bulbar and spinal-ganglion cells a further (secondary?) degeneration also extends toward the periphery into the corresponding nerve roots (hypoglossal, etc., spinal anterior roots) and nerve trunks. It is difficult to make out atrophied fibers in the peripheral nerves, and up to the present time they have not always been looked for with sufficient care, but it can scarcely be doubted that the affected motor fibers, which are processes of the atrophied ganglion cells, are likewise to be found in a condition of degeneration. Finally, the muscles show a considerable atrophy, as is plainly manifest even during the lifetime of the patient. Their volume is much diminished; many muscles (for details *vide infra*) finally almost wholly disappear, so that in their place there is little left but connective tissue and fat. In other muscles we find, besides a number of normal fibers still preserved, many very small fibers, showing various degrees of atrophy, with their transverse striation usually preserved, but more rarely indistinct. The nuclei of the sarcolemma are much increased, and the interstitial fat tissue is often, but not always, abundantly developed.

Although we may undoubtedly designate amyotrophic lateral sclerosis as, in the main, a primary degeneration of the motor tract, and especially of the pyramidal tract, yet in many cases the disease is not limited absolutely to this one system. Toward the lateral cerebellar tract (*KlS*) the degeneration is almost always very sharply limited—that is, the lateral cerebellar tract remains, as a rule, perfectly normal, but disease has repeatedly been found in Goll's columns. All these degenerations, last mentioned, have no marked clinical significance. They show only how other bundles of fibers may sometimes suffer under the same noxious influence as well as the pyramidal tract.

Our knowledge in regard to the questions as to where the degeneration begins, in what way it progresses, and what is primary and what secondary atrophy, is most uncertain. The anatomical examination gives only the result of the destruction, but it shows very few details of its course. Much may be inferred from clinical observation, but much work is still needed to attain perfectly clear knowledge. Perhaps different possibilities should be considered, which would explain many variations in the clinical course. At any rate the different portions of the system may be affected in varying sequence, and the disease may also extend with varying rapidity, but the destruction of the nerve fibers is always the primary process; the increase of the interstitial connective tissue and the slight changes in the vessels are a secondary, accidental process.

We know as yet hardly anything definite as to the causes of amyotrophic lateral sclerosis. For one class of cases we believe we may assume a congenital weakness of the motor system. The disease usually begins at the period between thirty-five and fifty, but not infrequently later. No special external causes of the disease have thus far been found. The male sex seems to be affected somewhat more frequently than the female.

Clinical History.—In all typical cases the clinical symptoms give a perfectly characteristic type of disease, limited strictly to the motor sphere, corresponding to the perfectly systemic anatomical lesions just described, The first signs of the disease almost always begin in the arm. The patient first notices (generally in one arm) a weakness and slight fatigue after motion and manual labor. The weakness of the arm gradually increases, and finally, usually some months later, involves the other arm. A wasting of certain muscles, which gradually increases and becomes more extensive, is now often noticed by the patient About six months or a year later, symptoms appear in the lower extremities. The gait becomes stiff and uncertain, the patient gets tired more easily, and quite a marked tremor of the legs often comes on, apparently spontaneously.

If we now examine the patient carefully, the type of the disease is usually perfectly plain. We notice, first in the upper extremities, a very pronounced and more or less extensive muscular atrophy. This is usually most marked where it also begins—namely, in the thenar and hypothenar eminences. The interossei are also plainly atrophied, and the muscles on the extensor side of the forearms, but the flexors of the hand and of the terminal phalanges of the fingers remain intact longer. In the upper arm the triceps and the deltoid are usually the most atrophied, and later, and to a less degree, the biceps and the muscles of the shoulder. We find a functional disturbance of the muscles—a paresis—corresponding to the degree of atrophy. The functional capacity depends upon how much muscle is left, and only with a complete disappearance of the muscle is there a complete loss of the corresponding motion, but a marked paresis can sometimes be noticed in muscles which are not yet much atrophied. Fibrillary muscular contractions are almost always to be detected in the affected muscles on careful observation. The electrical excitability of the muscular fibers still preserved is normal. The strength of contraction of the muscle irritated by the faradic current is therefore proportional to the amount of muscular substance still present. In the much-atrophied muscles the effects of irritation are finally very slight, and then we can always detect a distinct reaction of degeneration in the muscular fibers that are still left, especially in the ball of the thumb. We can scarcely ever make out with certainty a loss of excitability in the nerve trunk, probably because here a greater number of normal fibers are always present.

The examination of the tendon reflexes is very important. They are invariably much increased, even from the early stages of the disease. We obtain vigorous reflex contractions from a gentle blow on the tendons of the biceps and the triceps, and on the lower ends of the bones of the forearm. This increase of the tendon reflexes is extremely important in diagnosis, because it never occurs in this way in ordinary "progressive muscular atrophy"—that is, in that disease in which the degeneration extends merely from the muscles to the motor-ganglion cells in the anterior cornua, while the lateral motor tracts remain free (*vide infra*). In the later stages of the disease marked contractures in the arms and hands sometimes, but not always, develop. The sensibility of the skin and deeper parts, however, remains absolutely normal.

The first morbid symptoms usually develop in the lower extremities some months later than in the arms. The purely spastic symptoms are here remarkably prominent, while the muscular atrophy is late in its development, and is but slight. The legs become stiff, and oppose considerable muscular resistance to attempts at passive motion. The spastic symptoms are connected with the hypertonicity of the muscles (the increased muscle tonus) and the increased tendon reflexes. These symptoms depend mainly upon the great increase of the tendon reflexes. The patellar reflex is very vigorous, and we often find a marked and persistent ankle clonus. Generally true paretic symptoms are soon added to the spastic; at first, as a rule, in the flexors (shorteners) of the leg (i. e., in the flexors of the hip and knee, and the dorsal extensors of the foot). At every attempt to draw up the leg there is a marked contraction of the tibialis anticus, with a visible tightening of its tendon (so-called tibialis phenomenon). Stimulation of the sole of the foot generally produces reflex dorsal extension of the toe (Babinski reflex). [The Oppenheim reflex can also be obtained.—K.] The patient can still walk quite a distance, but the gait is, of course, difficult and laborious. The patient walks with short, slow, dragging steps—the spastic-paretic gait. Only in a few cases does a marked atrophy of the muscles appear in the early stages in the legs as well as in the arms. The sensibility of the skin and deeper parts is also perfectly preserved in the legs in amyotrophic lateral sclerosis. Disturbances in micturition are also entirely absent. The bowels may be somewhat costive, but they are otherwise normal.

After the condition has lasted for some time (a year or two) in this form —muscular atrophy and increased tendon reflexes in the upper extremities and spastic paresis in the lower—and has slowly grown worse, bulbar symptoms come on in the third and last stage of the disease. The speech gradually becomes more indistinct, and there is difficulty in swallowing. If we examine closely we find the lips atrophied, so that puckering the mouth, whistling, etc., are difficult. We also notice a decided atrophy of the tongue. Its surface is uneven, and we notice more or less marked fibrillary twitchings of single muscular bundles. The sensibility is also normal here. We sometimes find a vigorous masseter reflex [jaw-jerk] on striking the lower jaw, analogous to the increased tendon reflexes in the extremities. The other muscles of the face, the muscles of mastication and the ocular muscles, usually remain unaffected. The patient's intelligence is unchanged. The ready onset of marked mimetic emotional movements is sometimes striking. Many patients with amyotrophic lateral sclerosis break out into spasmodic laughter or weeping and wailing upon the slightest provocation ("impulsive laughing and crying"). This remarkable symptom, which also occurs in multiple sclerosis (page 329), is probably due to the disappearance of certain fibers which inhibit affective movements. The face is sometimes peculiarly distorted and broadened by a persistent tonic contraction of the facial muscles (see Fig. 187, page 395). In one case under our observation there were frequent attacks of severe spasm of the glottis with danger of suffocation.

If the patient have trouble in taking food, from difficulty in swallowing, the state of the general nutrition soon becomes worse. Respiratory disturbances are usually the final immediate cause of death, if an intercurrent disease, such as inhalation pneumonia, etc., does not previously put an end to the patient's melancholy condition.

Variations from this typical picture of the disease sometimes occur, but only as the sequence may vary in which the different symptoms come on or the different muscular regions are affected. If the anatomical lesion begins exclusively in the central motor neurons, there are at first purely spastic symptoms, either spastic rigidity of the lower extremities only or rigidity of the trunk and arms as well, and a distorted facial expression; and only later, when the second motor neurons are affected, is added atrophy of the muscles, at first usually either in the forearms and hands or in the bulbar region (tongue, lips, etc.). In other cases, however, the type of progressive muscular atrophy is from the first more prominent, and the spastic symptoms, if marked anywhere, appear only in the legs. In these cases the disease of the peripheral motor neurons is at first evidently greater than that of the central motor neurons; but even here the increased tendon reflexes point from the outset to an implication of the central motor neurons. Increased tendon reflexes, however, can, of course, occur only in those muscles whose ganglion cells in the anterior horns are not yet completely destroyed. The bulbar symptoms are wholly analogous to the other muscular atrophies; they are due to the degeneration of the nerve nuclei in the medulla. We shall become more fully acquainted with them later (*vide infra,* chapter on Progressive Bulbar Paralysis). Other differences in the clinical picture depend on whether the pyramidal tracts proper are exclusively involved, or whether the other motor anterolateral tracts are also affected. In the first case the spastic symptoms predominate; in the latter complete paralysis supervenes. I cannot forget a sad case of amyotrophic lateral sclerosis, in which the unfortunate patient, whose mind was perfectly normal, was finally able to move only the eyes, the diaphragm, and, to a slight extent, the head. The arms, legs, trunk, and tongue were completely paralyzed!

Diagnosis.—The diagnosis of the disease is rarely difficult. Its usually typical course, the muscular atrophy coexisting with increased tendon reflexes, the rigidity in other muscles, especially in the legs, the complete absence of sensory or vesical disturbances, and the final appearance of bulbar symptoms, are chiefly to be considered in diagnosis. Mistakes may arise from the fact that tumors or myelitis may for a long time have a similar localization, as in the gray matter of the cervical cord, and therefore provoke analogous symptoms. It is probable that in the past there was often confusion with syringomyelia (*q. v.*), but such mistakes may easily be avoided if we remember the sensory disturbances in the arms which are so very characteristic of this disease. The distinction between amyotrophic lateral sclerosis and the other primary diseases of the motor system will be self-evident from the description of these diseases which follows.

Prognosis.—The prognosis of amyotrophic lateral sclerosis must be regarded as absolutely unfavorable. The disease advances slowly but unceasingly, and usually leads to death in a few years.

We can therefore expect but slight results from **treatment.** At most we can perhaps check the advance of the disease by an electrical or gymnastic treatment, kept up with very great patience and perseverance.

3. SPINAL PROGRESSIVE MUSCULAR ATROPHY

Preliminary Remarks and Pathological Anatomy.—Few diseases of the spinal cord have been the subject of such different conceptions and interpretations as progressive muscular atrophy. The reason for this lies chiefly in the fact that its main symptom, the progressive atrophy of the voluntary muscles, may be found in many totally distinct diseases, and hence this has given rise to constant confusion and mistakes. If, at the present time, we read the older and even a part of the newer literature on this subject, we find everywhere a mixture of different cases, not at all of the same disease, and only the latest accurate clinical and anatomical methods of investigation have enabled us to bring at least some order out of this chaos.

Except for a few earlier observations, Duchenne and Aran, in 1849 and 1850, gave the first good description of progressive muscular atrophy. The French observers, therefore, at present speak of the disease, in distinction from other similar affections, as "*atrophie musculaire progressive, type Duchenne-Aran.*" A short time after, in 1855, Cruveilhier, on the ground of a positive lesion on autopsy, first pronounced the opinion that a disease of the gray matter in the spinal cord was to be regarded as the special anatomical cause of the disease. Since then a tedious dispute has been carried on, and even in part kept up to the present time, as to whether, in fact, the disease has its seat in the spinal cord, or in the muscles themselves—a dispute which was necessarily without results for a long time; the more because the actual pathological data were very scanty, and because the results of examinations often contradicted one another, owing to the confusion between different morbid processes which did not belong together at all.

At present we know that the great majority of progressive muscular atrophies certainly have their starting point in the muscles themselves (*vide infra,* progressive muscular dystrophy), but that there is also a rare but definitely established spinal form of progressive muscular atrophy. This is closely allied to amyotrophic lateral sclerosis, which we have just described; but although in amyotrophic lateral sclerosis the whole pyramidal motor tract may be degenerated, and the lateral pyramidal tracts in the cord are in particular always affected, in "spinal progressive muscular atrophy" the degeneration, as we have said, is confined to the peripheral motor neuron—that is, to that portion of the motor-conducting tract which extends from the ganglion cells of the anterior cornua to the muscular fibers themselves. The second central motor neuron—that is, the lateral pyramidal tract—remains perfectly normal. It is very improbable that this difference in the extent of the anatomical localization causes a fundamental distinction between the two diseases mentioned. The clinical distinctions due to the different anatomical localization are, however, of sufficiently marked prominence to justify, at least provisionally, a special limitation of amyotrophic lateral sclerosis on one side and spinal progressive muscular atrophy on the other, as two different clinical forms of the same morbid process.

The precise anatomical lesion in progressive (spinal) muscular atrophy is as follows:

In the spinal cord, particularly in the cervical cord, we find the anterior gray cornua very small; the ganglion cells have wholly or largely disappeared, and those remaining are atrophied; and the neuroglia is changed to a fine fibrous tissue, sometimes studded with spider cells; but the lateral columns, especially the pyramidal tracts—that is, the portion of the motor conducting tract central to the ganglion cells of the anterior cornua—are perfectly normal; the anterior roots and the affected motor fibers in the peripheral nerves, however, are atrophied, although in the nerve trunks the discovery of degenerated fibers mixed with many other normal fibers is not perfectly easy. In the muscles the atrophy is, of course, still more noticeable on post-mortem examination than on examination during life. The muscles most affected are reduced to small, pale, and flabby bundles, in which fat and connective tissue outweigh the proper muscular tissue. On histological examination we find, besides some muscular fibers which are still of normal diameter or even hypertrophied, all degrees of diminution and atrophy in the other fibers, so that finally all that seems to be left of many fibers is merely the empty sheaths of sarcolemma; but most of the diminished fibers retain their transverse striation to the last, and only occasionally do we see signs of granular (fatty?) and waxy degeneration of the muscular fibers, a splitting longitudinally and transversely, etc. The interstitial connective tissue is always hyperplastic, the muscular nuclei are considerably increased in number, and we often find an abundant deposit of fat between the fibers which are still preserved. The subcutaneous fat covering the atrophied muscles is also frequently developed to a marked degree.

So far the actual lesion. In comprehending it, the only difficulties arise from the questions as to the mode of development and as to the reciprocal dependence of the different disturbances. Is the atrophy of the anterior cornua to be regarded as primary, and the atrophy of the nerves and muscles as a secondary descending degeneration; or does the process begin in the muscles or nerve endings, and extend upward from them to the spinal cord; or, finally, do we have to do with an approximately simultaneous degeneration of the whole motor portion affected? These are questions to which at present no definite answer can be given. Many reasons seem to favor the belief that the degenerative process begins in the last terminal branches of the motor nerves, and from these ascends gradually to the spinal cord. But in some cases, at least, a primary atrophy of the motor-ganglion cells is also not improbable.

Ætiology and Clinical History.—Progressive muscular atrophy is an extremely slow and chronic disease from the beginning. The special causes are as little known as those of amyotrophic lateral sclerosis. Here, too, we shall probably have ultimately to fall back upon the hypothesis of conditions of congenital weakness in the affected systems of fibers. Hereditary and generic influences are often not to be detected, but they are sometimes undoubtedly present. We ourselves and other observers have repeatedly seen spinal muscular atrophy occur in several members of the same family. The disease sometimes begins in childhood (so-called family infantile type of spinal muscular atrophy of [Werdnig], J. Hoffmann, and others), and sometimes in youth, but often in more advanced age (from forty to fifty). Although, as we have said, a congenital weakness of the motor system is probably always to be regarded as the special cause of the disease, in some cases certain external conditions, especially persistent overexertion of the muscles in hard work, seem to favor its outbreak.

The disease begins, by far the most frequently, in the upper extremities, and especially, as it seems, in the right arm, but sometimes in the left, or in both arms at once. As a rule, it begins with an atrophy of the short muscles of the thumb and of the hypothenar eminence, which is accompanied by a corresponding disturbance of functions (see the accompanying figures, 170 to 172). Other symptoms—disturbances of sensibility, paræsthesia, or pain

—are usually entirely absent. The atrophy generally affects the abductor pollicis brevis and the opponens first, and then the flexor brevis and the adductor. We notice very early the characteristic sinking in and flattening of the ball of the thumb, and the abnormal position of the thumb, which is persistently approximated to the second metacarpal bone—the "ape hand." It becomes constantly harder and finally impossible to oppose the thumb to the

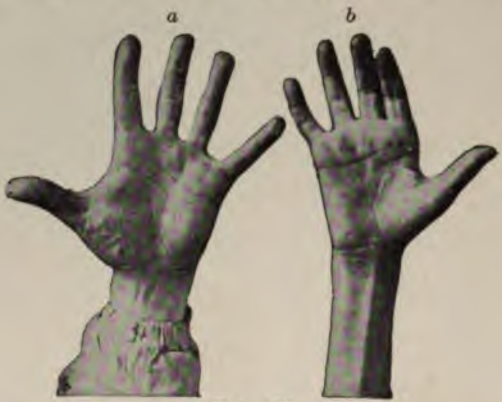

FIG. 170.

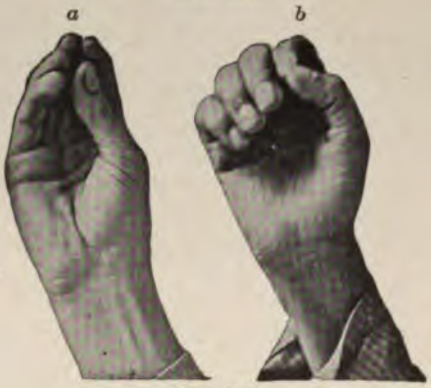

FIG. 171.

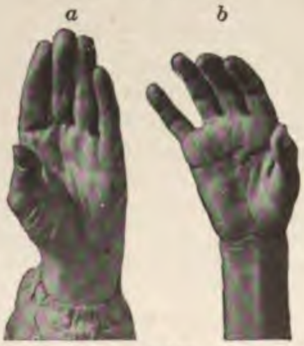

FIG. 172.

FIGS. 170–172.—Atrophy of the small muscles of the hand, especially of the thenar and hypothenar muscles, in spinal progressive muscular atrophy. *a.* Hand of a healthy man. *b.* Atrophied hand. (Erlangen Medical Clinic.)

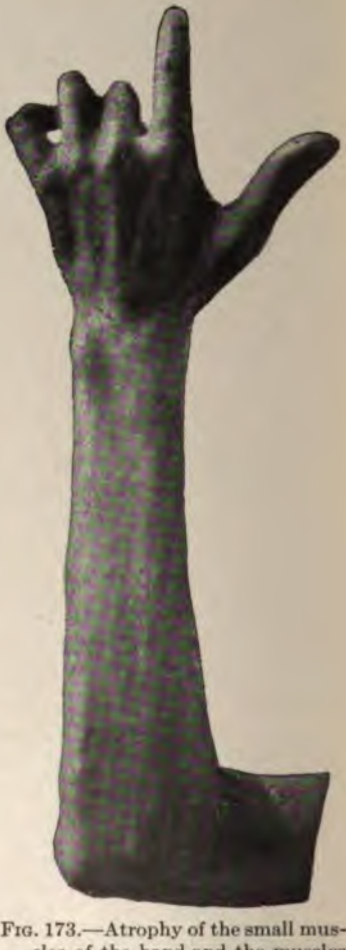

FIG. 173.—Atrophy of the small muscles of the hand and the muscles on the extensor side of the forearm in spinal progressive muscular atrophy. The last three fingers can no longer be extended. (Personal observation.)

other fingers (performed by the flexor brevis for the index and middle fingers and by the abductor brevis and opponens for the ring and little fingers). At the same time, or a little earlier or later, the interossei begin to atrophy, as is recognized by the sinking in of the interosseous spaces and the increasing incomplete extension of the terminal phalanges of the fingers. The atrophy of the lumbricales causes a visible flattening of the hollow of the hand. If the disturbance in the function of the interossei have reached a certain degree, the same claw-like position of the fingers appears, as we have already learned to recognize in ulnar paralysis (see Fig. 173), due to the contracture of the antagonist of the interossei, the extensor communis digitorum.

In the further progress of the disease the atrophy either extends to the muscles of the forearm, or, what is not at all rare, it jumps over these and attacks the muscles of the shoulder, usually the deltoid first. In the forearm it is usually the muscles on the extensor side which are first attacked, the abductor and extensor longus pollicis, the extensors of the wrist and fingers, and only later the supinators, flexors, etc. In the upper arm the deltoid almost always atrophies first, and then the biceps, while the triceps may remain intact somewhat longer. There are also cases in which the atrophy begins in the deltoid and the muscles of the upper arm, while the small muscles of the hands and fingers are not affected until later. The muscles of the trunk are usually added to the list quite late—the muscles that extend the head and keep it erect, so that the head sinks forward—the trapezii, the infraspinati and supraspinati, the rhomboidei, the latissimi dorsi, the pectorals, etc. (see Figs. 174 and 175). The disturbance of function caused by the atrophy of all these muscles is readily apparent from what was said in the chapter on the different forms of paralysis. In advanced cases the arms hang down laxly by the sides of the trunk. Only with the greatest difficulty, if at all, can the patient do anything with them—dress or un-

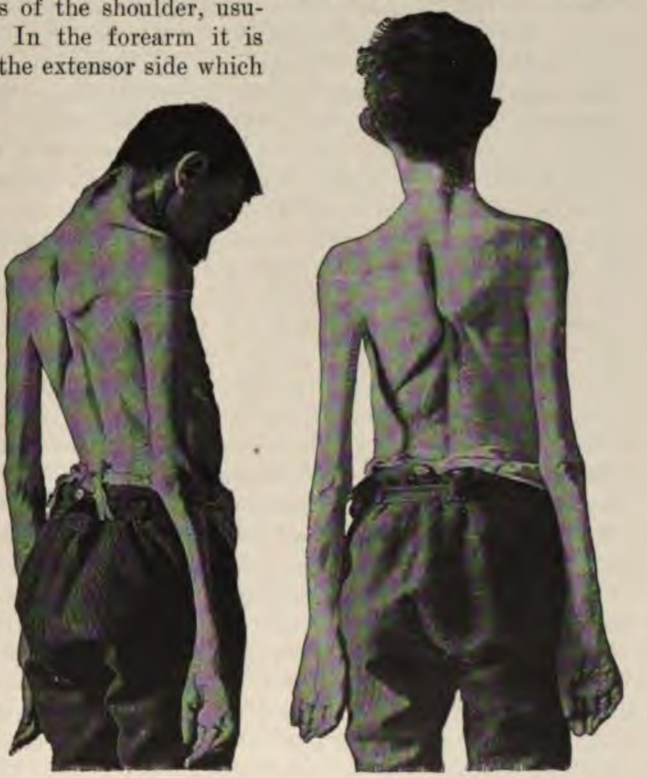

FIG. 174. FIG. 175.

FIGS. 174 and 175.—Spinal progressive muscular atrophy. (Personal observation.)

dress himself; but he sometimes learns to help himself, at least in some degree, by "throwing" movements, by motions of the trunk, by using his mouth in taking hold of things, etc. Quite rarely the atrophy finally attacks the muscles of the neck. The severest respiratory disturbances, and finally a fatal termination, may be brought about by an implication of the diaphragm and other muscles of respiration.

The time which elapses before the gradual appearance of the more marked disturbances of function is almost always very long. Years may elapse before the atrophy extends from the small muscles of the hand to the other muscles of the arm. In the muscles of the legs the first signs of atrophy almost always develop very late, if at all. The arms are often perfectly useless when the patient can still walk for hours. Of course there are some exceptions to this rule, and then the muscles of the lower leg (peronei), especially, seem to be first affected.

Besides the atrophy, and the loss of function that runs parallel to it, we must mention some other changes in the muscles. The fibrillary muscular twitchings are often very striking. A constant tremor and wave of the muscle may be provoked by them. In some cases they are weak and they can be detected only by special attention. They usually become vigorous if one irritates the muscle mechanically by a blow. It is of the greatest diagnostic importance to determine positively the presence of a true fibrillary muscular tremor (not to be confused with the muscular tremors of neurasthenics, the action of cold in patients when undressed, etc.), since this symptom almost invariably points with certainty to a disease of the spinal-motor ganglion cells. The electrical excitability of the diseased muscles varies, inasmuch as it depends entirely upon the number of normal fibers still preserved in the muscle. Since the atrophy affects only one fiber after another, the faradic and galvanic excitability decrease gradually, and are lost completely only when the greatest part of the muscle is destroyed. On careful testing we can then, however, almost always make out a decided galvanic reaction of degeneration in single muscles that are much diseased, especially in the form of the so-called partial reaction of degeneration—the excitability of the nerves is retained, but the contractions in the muscles themselves seem very slow, and the anodic closure contractions (*AnSZ*) predominate (see page 219).

In many cases, as has been mentioned, there is an increase of the fatty tissue (which is always of moderate degree) at the same time with the atrophy of the muscular substance, and this often makes it very hard to judge of the atrophy; but the loss of function in the muscles, the diminished electrical excitability, and the peculiar soft feeling which atrophied muscles have when covered with fat, reveal the true condition of things. There are no trophic disturbances in the skin. Almost all the former statements in regard to this refer probably to the confusion between progressive muscular atrophy and similar forms of disease, especially syringomyelia. The coldness and cyanosis sometimes seen in the hands is due to the fact that the arms constantly hang down and to the lack of any muscular movement.

The examination of the tendon reflexes is of great diagnostic importance. While they are invariably decidedly increased in the upper extremities in amyotrophic lateral sclerosis, they are only feebly manifest or entirely absent in genuine progressive muscular atrophy, a condition which is easily explained by the atrophy of the motor-ganglion cells belonging to the reflex arc, or by the atrophy of the centrifugal motor fibers. Since no degeneration of the lateral pyramidal tract precedes this atrophy, it goes without saying that the muscular rigidity, associated with paresis and increase of the tendon reflexes, so characteristic of amyotrophic lateral sclerosis, is also absent. Muscular weakness and muscular atrophy are completely parallel in spinal progressive muscular atrophy. In the lower extremities the patellar reflex is retained as long as the disease spares the legs, but it is not increased. If the atrophy involve the legs, the patellar reflex is usually lost.

In distinction from all these manifest disturbances in the motor region, the sensibility of the skin and deeper parts remains perfectly preserved. A careful test of the sensibility is therefore indispensable to avoid mistaking it for similar morbid conditions, such as syringomyelia. There are also never any morbid symptoms on the part of the bladder or rectum.

In many cases the affection finally invades the muscular region innervated from the medulla; the symptoms of "progressive bulbar paralysis" (*vide infra*) are added to those of progressive muscular atrophy. This combination of spinal and bulbar disease, as we have previously shown, is a usual occurrence in amyotrophic lateral sclerosis, even when the disease has lasted a comparatively short time. In genuine progressive muscular atrophy, however, the bulbar symptoms usually develop, if at all, only after the disease has gone on for years. Then the speech begins to be indistinct from the atrophy of the tongue, swallowing is difficult, and the patient finally succumbs to increasing inanition or to respiratory disturbances. In their principal characteristics the muscular atrophy of the extremities and the bulbar symptoms, as we have said, are precisely analogous phenomena, inasmuch as the nerve nuclei in the medulla have precisely the same significance for the muscles of the tongue, the pharynx, and the face, as the anterior gray cornua of the cord have for the muscles of the trunk and the extremities. In many cases, however, bulbar symptoms do not develop at all, since the patients die early of some intercurrent disease, or the fibers of the bulbar region are not involved in the morbid process; but the whole process may begin, as we shall see later, in the region of the bulbar nerves as "bulbar paralysis," while the spinal muscular atrophy does not come on in the arms until afterwards.

Diagnosis.—The diagnosis of spinal progressive muscular atrophy can easily be made if we confine ourselves strictly to the definition of the disease, and do not confuse it with other affections in which the muscular atrophy is only one symptom which may have an entirely different origin—muscular atrophies in extensive diffuse myelitis, in tumors, in the formation of cavities in the cord, in multiple neuritis, as a result of articular affections and injury, etc. We should consider especially the typical course of the affection in most cases of genuine progressive muscular atrophy, its beginning in the upper extremities, the small muscles of the hand, or, more rarely, the muscles of the shoulder and upper arm, its slow advance, the peculiar "individualization" of the atrophy—that is, the affection of some muscles while other neighboring muscles remain completely normal; the special form of atrophy recognizable by fibrillary contractions and electrical reaction of degeneration; and, finally, the absence of all disturbances of sensibility or of the sphincters. Progressive muscular atrophy is doubtless nearly allied to amyotrophic lateral sclerosis, but the latter is distinguished by its more rapid course, and especially by the increase of the tendon reflexes due to the affection of the lateral columns and the corresponding appearance of spastic symptoms. The differential diagnosis between the spinal and the myopathic (juvenile) muscular atrophy (muscular dystrophy), which is also perfectly certain at present in most cases, will be described later.

Prognosis.—The prognosis of progressive muscular atrophy is to be regarded as absolutely unfavorable. The disease appears comparatively benign only from the fact that it is apt to advance very slowly, since it may last for ten or fifteen years, or even longer. As we have already said, the fatal termination at last arises from some intercurrent disease, or as a result of the final appearance of dangerous bulbar symptoms, paralysis of deglutition and respiration.

Treatment.—The results of treatment are accordingly very slight. An electrical treatment, continued for months and years with very great persistence, can alone produce à little improvement or check the advance of the atrophy somewhat. Temporary improvement may also sometimes be attained by methodical massage of the muscles and a rationally conducted gymnastic treatment. In other respects the treatment must be symptomatic. [Gowers claims that daily subcutaneous injections of strychnin, gr. $\frac{1}{100}$ to $\frac{1}{40}$ (gm. 0.0005 to 0.002), often arrest the disease.—K.]

4. NEUROTIC MUSCULAR ATROPHY

(Type Charcot-Marie. Peroneal-Forearm Type of Progressive Muscular Atrophy)

This name is given to a form of progressive muscular atrophy which has become known through many recent observations (Charcot and Marie, Tooth, J. Hoffmann, and others), and which is manifested by several very characteristic clinical features. The disease is often a pronounced hereditary and family affection, which can sometimes be traced through four or five generations, but, on the other hand, it also occurs in members of the same generation without being present in the antecedents, and individual cases have also repeatedly been observed in which there was apparently no hereditary predisposition. The first symptoms of the disease usually appear in childhood, but some cases have also been observed in later life. There is no essential difference between the two sexes.

Next to the conditions mentioned (beginning in childhood, generic occurrence), the most noticeable feature is the very regular localization of the muscular atrophy (see Fig. 176). The atrophy almost always begins symmetrically in the portions of the extremities most distant from the trunk—that is, in the hands and forearms and the feet and lower legs. The lower extremities are often affected for a longer or shorter time (even for years) before the upper, but the contrary sequence may also occur. Since the atrophy of the small muscles of the feet causes symptoms that attract little attention (a claw-like position of the toes), the disease may often remain unnoticed until the peronei, the tibiales antici, and the long extensors of the great toe are involved. The gait then becomes impaired from the dropping of the toes and shows all the characteristic peculiarities of bilateral peroneal paralysis (*q. v.*). In older cases there often develops on both sides a complete talipes equinus or varo-equinus. As the atrophy progresses, the muscles of the calves gradually become affected, and finally the muscles of the thigh as well. Atrophy and paresis of the muscles always go hand in hand. The same is seen in the upper extremities. Here the small muscles of the hand, the interossei and thenar and hypothenar muscles, atrophy first, just as in spinal muscular atrophy and amyotrophic lateral sclerosis. The fingers therefore assume the claw position; the movements of the thumb become more and more impaired, so that it lies adducted against the hand. The atrophy then extends to the forearms, where the extensors are earlier and more markedly affected than the flexors. Later in the course of the disease, when the forearms and hands are already greatly emaciated, we can usually discover the beginning of atrophy in the muscles of the shoulder and upper arm. The muscles belonging to the trunk resist the morbid process the longest. The facial muscles also are usually spared, although manifest changes may finally occur even in them, but no disturbances have thus far been observed in the domain of the bulbar nerves. Finally, we may mention the observations reported by Hänel where the disease was traced in many members of one family through four generations and was always confined to the upper extremities.

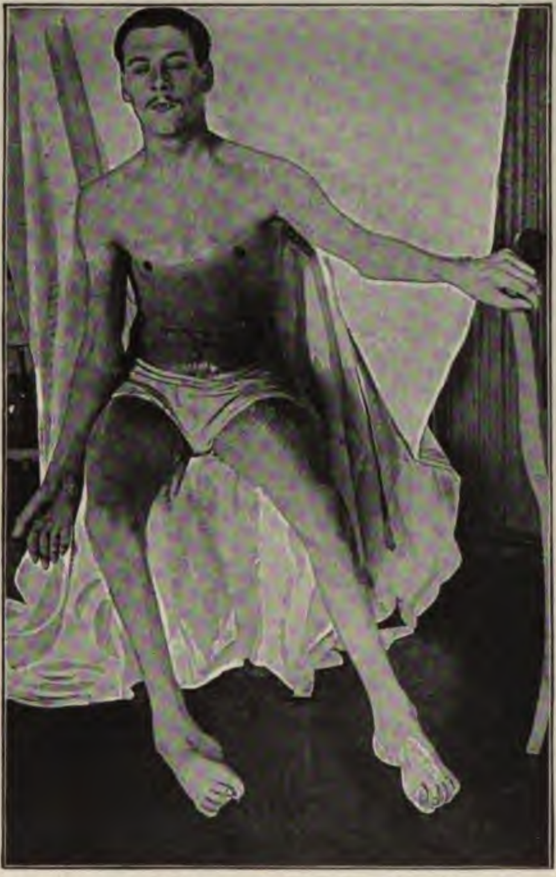

FIG. 176.—Neurotic muscular atrophy. The disease came on in almost the same fashion in three members of one family. (Personal observation.)

It is of importance for our comprehension of the whole disease that the atrophying muscles show fibrillary contractions, usually slight, only exceptionally lively, and that we can invariably find the most marked changes in the electrical excitability of the muscles and their corresponding nerves. The nerves soon show a marked diminution or even a total loss of galvanic and faradic excitability. In the muscles we find either the same conditions or a pronounced galvanic reaction of degeneration. It is a very interesting fact that, on electrical examination, we can often detect manifest changes in excitability, just as in lead paralysis, in muscles which apparently show no atrophy or paralysis at all.

The mechanical excitability of the muscles is also diminished. The tendon reflexes of the affected muscles are wholly absent or much diminished. They are never increased. The sensibility is never much affected, but it is often not perfectly normal. Paræsthesiæ and painful sensations in particular are often present, and there is sometimes a manifest but slight blunting of the cutaneous sensibility. The functions of the bladder and rectum remain normal.

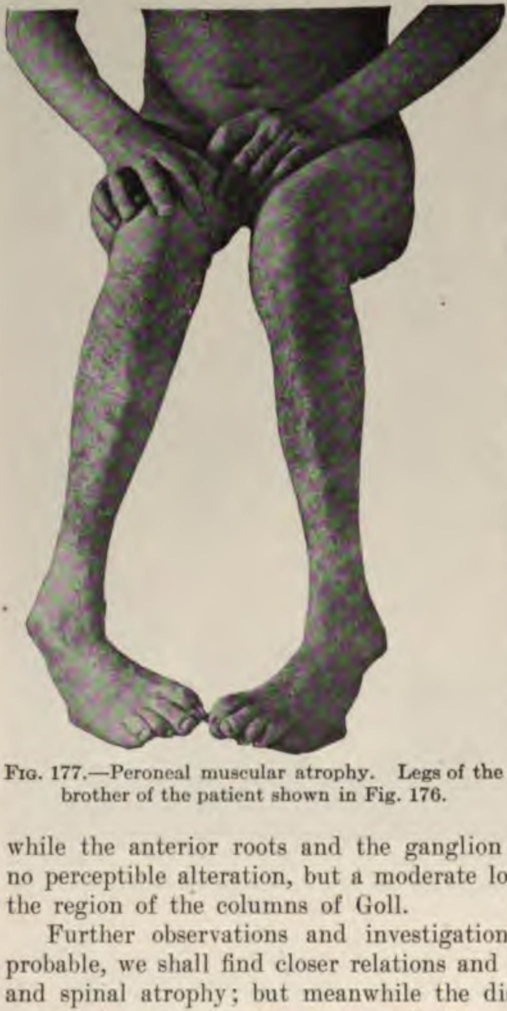

FIG. 177.—Peroneal muscular atrophy. Legs of the brother of the patient shown in Fig. 176.

From the clinical facts just described the conclusion may be drawn that the form of muscular atrophy under discussion must have a neurotic or a spinal origin. The assumption of a neurotic origin is favored not only by the results of the electrical examination and the occurrence of pain and slight sensory disturbances, but also by the results of the anatomical investigations thus far reported, although they are, of course, still few in number. According to these, the disease seems to be confined mainly to the muscles and the peripheral motor nerves, while the anterior roots and the ganglion cells in the anterior horns show no perceptible alteration, but a moderate loss of fibers is sometimes found in the region of the columns of Goll.

Further observations and investigations must teach us whether, as is probable, we shall find closer relations and transition forms between neurotic and spinal atrophy; but meanwhile the disease just described is a form of progressive muscular atrophy so well characterized and so readily recognizable in its clinical aspect that a separate description of it is undoubtedly warranted.

[Dejerine and Sottas have described, under the name of "hypertrophic interstitial neuritis," an affection similar to neurotic muscular atrophy, which also begins in early life and may affect several members of the same family. The atrophy usually begins in the legs, being most marked at the extremity and gradually extending upward toward the trunk. The arms, and sometimes the face, are affected later in the same way. The reaction of degeneration can be found in the affected muscles. There is a very pronounced enlargement of the nerve trunks, which can often be felt and sometimes even seen. This is the characteristic feature of the disease. In addition there may be well-marked kyphoscoliosis, loss of tendon reflexes, inability to stand with the eyes closed, Argyll Robertson pupils, or sluggish pupils with myosis, hypæsthesia and delayed sensation, very severe shooting pains, ataxia, and occasionally intention tremor, scanning speech, and slight exophthalmus. The sphincters are never involved. The trouble is very slowly progressive. Autopsies have shown, besides a very extensive and pronounced neuritis, lesions in the anterior cornua and posterior columns of the cord. The prognosis is unfavorable, and the treatment is like that of other forms of atrophy.—K.]

5. PROGRESSIVE MUSCULAR DYSTROPHY

(The Myopathic Juvenile Forms of Progressive Muscular Atrophy)

The so-called purely myopathic muscular atrophies are much commoner than the forms of muscular atrophy thus far described, in which, besides the wasting of the muscular fiber, there is a more or less extensive atrophy of the motor nervous system. Myopathic muscular atrophy is the name given to all those forms in which anatomical investigation has thus far found atrophic processes, not in the cord or in the peripheral nerves, but exclusively in the muscles themselves. The old, long contest whether the "seat" of progressive muscular atrophy was to be looked for in the spinal cord or in the muscles themselves, has thus at last been decided—that each of these hypotheses was correct for certain cases. Thanks to persistent clinical and anatomical investigations, we are now in a position to distinguish, easily and positively for the most part, the spinal (and neurotic) from the myopathic cases even during the patient's life. Of course many facts even now seem to indicate that there is no very strict division, and that here also transition forms may occur which can be fully understood only by a still more accurate insight into the nature of the morbid process in question.

In spite of many common peculiarities, the individual cases of myopathic muscular atrophy differ in many respects from one another. These differences relate either to the localization of the muscles chiefly or at least first affected, or to the special variety of change in the muscles (simple atrophy or atrophy with a large interstitial development of fat). These differences were the reason why it was formerly believed that several forms of disease could be recognized, whereas in reality we are dealing only with different types of the same disease. These varieties, of course, have so much individuality and so constantly recur in the same way that we are perfectly justified in differentiating them from a clinical standpoint; but, on the other hand, there are so many transitions between the different types that their intimate relationship is undoubted. Erb has therefore been perfectly correct in proposing to group together all these different types under one common name—*progressive muscular dystrophy*—in order to attest their kinship. We can then distinguish the different subtypes by special terms.

Before we proceed in what follows to describe these different subtypes in greater detail, we would first mention the most important points which are common to all forms of myopathic muscular atrophy—that is, of so-called muscular dystrophy. They are as follows: (1) All forms of dystrophy occur generically or hereditarily. We therefore see the disease most frequently in brothers and sisters, or more rarely in members of the same family in different generations. On the other hand, sporadic cases are, of course, not uncommon. If the disease occur in members of the same family it usually assumes the same form in each of them; but different forms may exceptionally occur in one family—a circumstance strongly suggesting an intimate relation between the different types. (2) The first symptoms of the disease almost always appear in youth, sometimes in childhood, sometimes not until puberty. Only in rare cases does the disease begin between thirty and forty or even later. It is worthy of note that certain forms usually begin in childhood ("infantile" form with implication of the facial muscles, pseudo-hypertrophy), and other forms only at puberty ("juvenile" form with implication of the shoulder and upper-arm muscles). (3) The localization of the muscular atrophy is extremely irregular. The different forms or types are, of course, distinguished by their special localization; but, as the disease advances, or in the frequent transition cases, it often happens that other muscular regions besides those chiefly affected are also involved to a lesser degree. It thus appears that in all forms it is finally the same muscular regions which are chiefly affected with atrophy, while, on the other hand, in all forms certain muscular regions, with scarcely an exception, remain intact. The groups of muscles most exposed to the disease are: (*a*) Facial muscles, as far as they are innervated by the facial nerve, especially the orbicularis palpebrarum and orbicularis oris. (*b*) Muscles of the shoulder girdle: pectoralis, latissimus dorsi, serratus anticus major, rhomboidei, trapezius. (*c*) Muscles of the upper arm: biceps, brachialis anticus, supinator longus, triceps. (*d*) Long extensors of the back (erectores spinæ). (*e*) Pelvic muscles (glutæi) and muscles of the thigh. Other muscles, however, are almost always spared in muscular dystrophy: (*a*) Deltoids, supraspinati, and infraspinati. (*b*) Forearm muscles, and especially the small muscles of the hand and fingers. (*c*) Lower-leg muscles, especially the gastrocnemius and the small muscles of the foot. (*d*) Tongue and muscles of deglutition. All these statements are not absolutely without exception, but they show the usual rule, which must depend upon the essential nature of the disease. (4) The anatomical changes in the muscles are essentially the same in all the different forms of dystrophy. They consist first of the most varied changes in volume of the individual muscular fibers. Many fibers show an atrophy which progresses finally to complete disappearance, while, on the other hand, so-called hypertrophic (hypervoluminous) fibers are also almost always to be found. Erb suspects that the hypertrophy is the first stage of the disease of the fiber, and that the atrophy follows this later. Besides these changes in volume, we see regularly an evident increase in the muscle nuclei and a considerable increase in the interstitial connective tissue. If there be pronounced pseudo-hypertrophy the interstitial tissue is permeated with an abundant accumulation of fat cells. In the muscular fibers themselves the transverse striation is almost always preserved to the last, and we never find granular or fatty, so-called degenerative destruction in any great degree. The peripheral motor nerves and the motor-ganglion cells in the anterior horns—that is, the peripheral motor neurons—remain perfectly normal. Two clinical facts, which are very important in diagnosis, are associated with this circumstance: (*a*) In all forms of dystrophy the fibrillary contractions in the diseased muscles, which are so common in spinal atrophies, are almost invariably absent. (*b*) Distinct electrical reaction of degeneration is almost never found in dystrophy (in contrast to the spinal atrophies). Since the disease is also a motor-system disease, like all the other forms of progressive muscular atrophy, it is self-evident that in dystrophy the sensibility, the functions of the bladder, etc., remain perfectly normal.

Since we have mentioned the most important features common to all forms of dystrophy, we will pass to the description of the chief individual clinical types. We would emphasize the fact once more that transition forms between these types are by no means rare.

1. PSEUDO-HYPERTROPHY OF THE MUSCLES (*Lipomatosis luxurians muscularis progressiva* of Heller; *Atrophia musculorum lipomatosa* of Seidel).—Pseudo-hypertrophy is the rarest form of muscular dystrophy, but it was the first to become accurately known, on account of its striking aspect. It was quite fully described in Germany by Griesinger (1864), while in France Duchenne gave in 1868 a very complete clinical description of it. In 1866 M. Eulenburg and Cohnheim had already shown, by the careful anatomical examination of a case, that the nervous system remained perfectly normal, a statement which was confirmed by all subsequent investigations (Charcot, F. Schultze, and others).

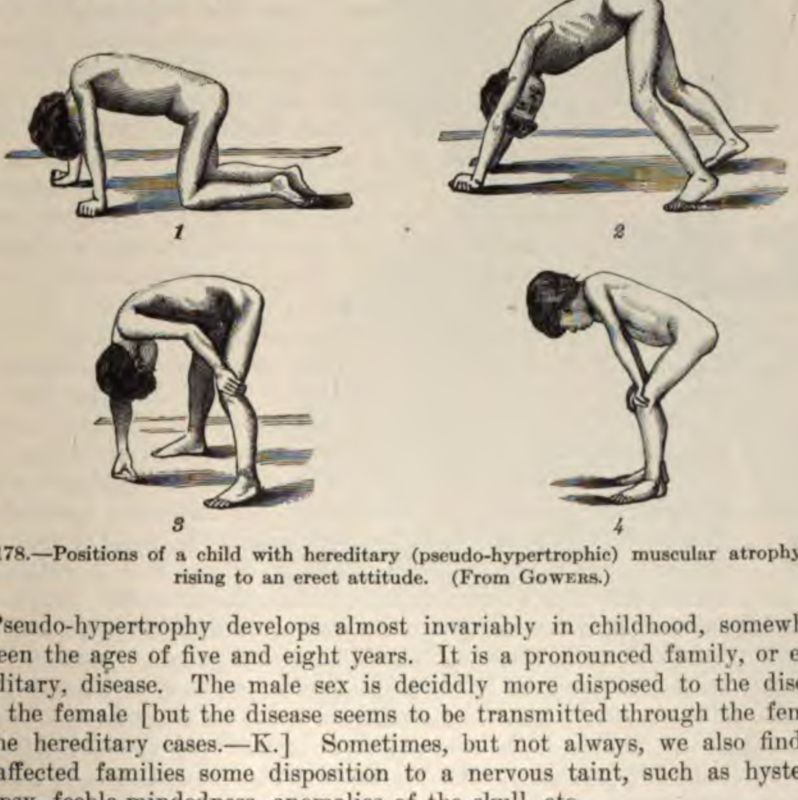

FIG. 178.—Positions of a child with hereditary (pseudo-hypertrophic) muscular atrophy, on rising to an erect attitude. (From GOWERS.)

Pseudo-hypertrophy develops almost invariably in childhood, somewhere between the ages of five and eight years. It is a pronounced family, or even hereditary, disease. The male sex is deciddly more disposed to the disease than the female [but the disease seems to be transmitted through the female in the hereditary cases.—K.] Sometimes, but not always, we also find in the affected families some disposition to a nervous taint, such as hysteria, epilepsy, feeble-mindedness, anomalies of the skull, etc.

The disease begins gradually and almost always without a special exciting cause. The parents notice that the child, who had been previously perfectly well and strong, becomes insecure on his legs, so that he can no longer jump or go upstairs as well as he used to do. This points to the first characteristic peculiarity wherein pseudo-hypertrophy differs from spinal progressive muscular atrophy. It begins, with rare exceptions, in the muscles of the trunk, especially in the muscles of the back and pelvic muscles (glutæi), and in the muscles of the lower extremities, especially in those of the thigh. While the arms and hands are still perfectly normal, walking constantly grows more and more difficult, and the gait very soon assumes so characteristic a type that from this alone the diagnosis can often be made at the first glance. The gait becomes waddling (chiefly on account of the weakness of the glutæi medii), the belly appears very prominent, the vertebral column is arched forward in the lumbar region in marked lordosis, and the whole upper part of the body is balanced on the legs. The legs are raised slowly and with difficulty, and the toes usually droop from paresis of the dorsal extensors. The child's movements, when he tries to raise himself from the floor or pick up any object, are very characteristic, and are alike in almost all cases, as a result of paresis of the extensors of the thigh and leg (glutæi and extensores cruris). Since it is impossible to raise the trunk, the child usually first gets on all fours, and then gradually straightens himself up ["climbs up his thighs"] by supporting his arms on his knees (see Fig. 178). Later on disturbances of motion appear in the upper extremities also, and in general they are very similar to those to be described more fully in the next type.

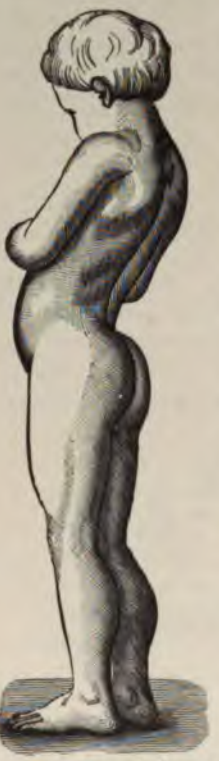
FIG. 179.—Pseudo-hypertrophy of the muscles. (From DUCHENNE.)

If we inspect the patient more closely we shall usually find at the first glance an extraordinary increase in the volume of single muscles (see Fig. 179). The calves are disproportionately thick, and sometimes the thighs also; the arms are affected later, especially the deltoids, the triceps, etc. This increase of volume is caused by an abnormal interstitial development of fat—"pseudo-hypertrophy." Hence, the muscles do not feel firm, but soft and spongy. It is by no means rare, however, that, besides the pseudo-hypertrophy in some muscles, a genuine atrophy develops in others, with a pronounced loss of substance and without any coexisting development of fat. This is seen especially in the upper extremities in the groups of muscles previously mentioned. Finally, there seems to be in addition even a genuine muscular hypertrophy. In several cases we have seen a marked increase in volume in the muscles of the calves, which were capable of quite an extraordinary display of strength. In such cases, however, there is, probably, in our opinion, a sort of compensatory hypertrophy, since the muscles that are still able to work are exerted excessively.

Fibrillary twitchings of the muscles are scarcely ever seen. Electrical examination shows a diminution of excitability corresponding to the atrophy and to the increased deposit of fat, but never reaction of degeneration. The sensibility remains perfectly normal, and also micturition and defecation. The patellar reflex is sometimes absent, because the quadriceps is involved. It is noticeable that the skin, especially in the legs, very often shows a peculiar bluish marbled coloring. Bulbar symptoms apparently never occur. The intelligence in most cases is perfectly normal, but exceptionally children with hereditary muscular atrophy also show manifest signs of mental or even of moral weakness.

The disease advances very slowly but without remissions. Finally, the patient cannot walk at all; he is confined to the bed, and becomes more and more helpless. Death usually ensues from some intercurrent disease, but sometimes from insufficiency of the respiratory muscles.

2. INFANTILE ATROPHIC FORM OF MUSCULAR DYSTROPHY WITHOUT AND ESPECIALLY WITH IMPLICATION OF FACIAL MUSCLES.—Simple atrophic forms of progressive muscular wasting develop far more frequently in early childhood than pseudo-hypertrophy. They may be combined with pseudo-hypertrophy, as we have said above, especially in the form where there is lipomatosis in the lower extremities and simple atrophy in the shoulders and arms; but what gives the simple atrophic infantile form a special stamp is the comparatively frequent implication of the facial muscles. These muscles may not be involved, and, on the other hand, they may be affected in rare instances in the juvenile form as well, but marked atrophy of the facial muscles is a peculiarity chiefly of the infantile atrophic form.

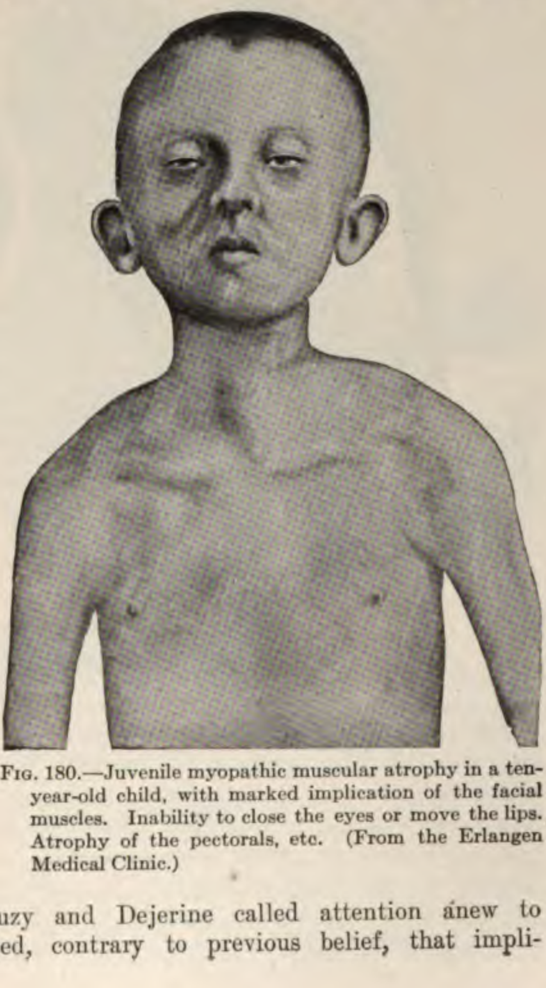

FIG. 180.—Juvenile myopathic muscular atrophy in a ten-year-old child, with marked implication of the facial muscles. Inability to close the eyes or move the lips. Atrophy of the pectorals, etc. (From the Erlangen Medical Clinic.)

Duchenne had already observed that in children there is a form of muscular atrophy which begins in the muscles of the face; but his statements were almost forgotten until recently Landouzy and Dejerine called attention anew to this subject, and demonstrated, contrary to previous belief, that implication of the facial muscles is by no means an unusual symptom. If the atrophy is already plainly pronounced, characteristic symptoms ensue: the eyes can no longer be completely closed, and the movements of the mouth in whistling, laughing, and talking become impaired. From the sinking in of the cheeks, the drooping of the under lip, etc., there develops a very characteristic type of countenance ("*facies myopathique*"), by which the practiced eye can recognize the affection almost at once (Fig. 180). There may finally ensue complete rigidity and immobility of the face. At the same time or somewhat later, as a rule, other muscular regions may be affected, and again, as in other forms, chiefly the shoulder muscles, the muscles of the upper arms, the extensors of the spine, and the muscles of the pelvis and thigh. The child's gait becomes as waddling as that of children with pseudo-hypertrophy, which we have just described. The spine has a lordotic curvature, the child rises from the floor by the aid of the arms (Fig. 178), etc. The sequence in which the different muscles are attacked by the atrophy is not always the same. The facial muscles, for example, may often be attacked later than the muscles of the shoulder, upper arm, and trunk. In order to detect the slighter degrees of implication of the face, it is advisable to have the patient close his eyes when the head is bent backward; we can then readily detect the fissure left between the eyelids (insufficiency of the orbicularis palpebrarum).

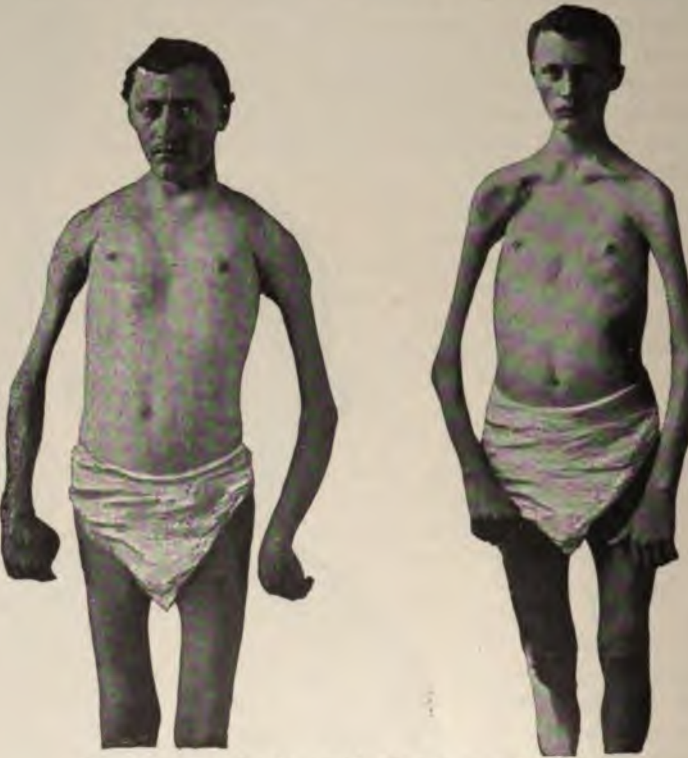

FIG. 181.—Two brothers with juvenile muscular atrophy.
(Personal observation.)

We can also notice in the early stages that the patient cannot protrude the lips to whistle.

The muscles of mastication, the internal ocular muscles, and the muscles of the forearm and hand almost always remain normal. A slight persistent contracture of the biceps is sometimes present and is characteristic. Fibrillary contractions and reaction of degeneration are almost always absent.

3. THE JUVENILE FORM OF DYSTROPHY.—The peculiarities of this form were first carefully studied by Erb. It usually does not begin until after actual childhood, in the years of puberty or sometimes later (between twenty and forty). It occurs sporadically occasionally, but it is also frequently hereditary or generic (that is, occurring in families), and the girls of the family are often attacked by this form, while the infantile form is seen especially in boys. The disease is also, like pseudo-hypertrophy, sometimes seen to begin in the pelvic muscles and the legs, but more often the shoulders and upper extremities are first attacked. There is also a remarkable regularity in the choice of the muscles attacked, as has been mentioned above. According to Erb, the following muscles are almost constantly diseased in the trunk and the upper extremities: the pectoralis major and minor, the trapezius, the latissimus dorsi, the serratus anticus major, the rhomboidei, the sacro-lumbalis and longissimus dorsi, and later the triceps. The following, however, almost always remain normal; the sterno-mastoid, the levator anguli scapulæ, the coraco-brachialis, the teres major and minor, the deltoid, the supraspinatus and infraspinatus, and, as must be mentioned especially in distinction from spinal muscular atrophy, the small muscles of the hand. The muscles of the forearm, too, except the supinator longus, remain intact for a long time, if not permanently; but exceptions also occur: for example, the accompanying illustration (Fig. 181) shows two brothers with juvenile muscular atrophy which in other respects is characteristic, but the muscles on the extensor side of the forearm are also completely atrophied. In the lower extremities the atrophy attacks chiefly the glutæi and the quadriceps, more

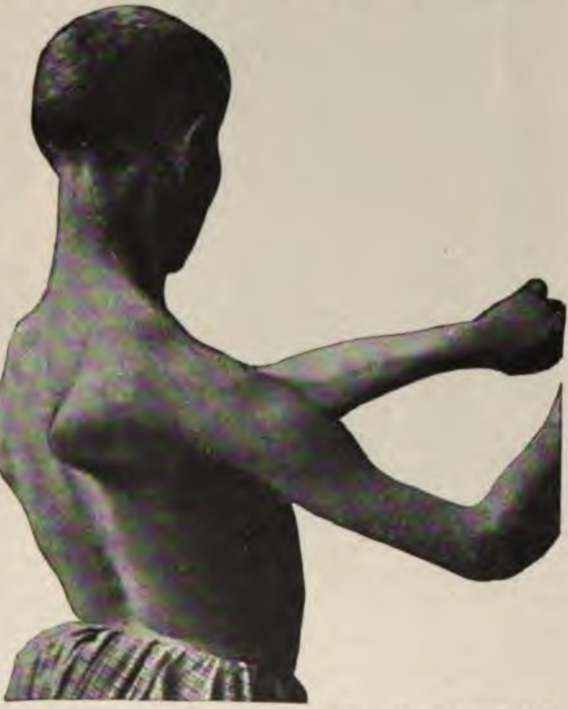

FIG. 182.—Juvenile muscular dystrophy. Prominence of the scapulæ when the arms are raised in consequence of atrophy of the serratus and rhomboidei. (Personal observation.)

rarely the peronei and the tibialis anticus, while the sartorius and the muscles of the calf are usually wholly exempt. Fibrillary twitchings in the affected muscles are generally absent, and there is hardly ever any reaction of degeneration.

The disturbances of function due to this condition are self-evident, so that a full description of them may be omitted. The arms usually suffer first, as we have said. The marked projection of the scapulæ, from the paralysis of the serratus, is especially characteristic (Fig. 182). If we try to take the patient under the shoulders and lift him, the shoulders are perfectly limp. They are therefore immediately raised so high that the head comes between the shoulders ("loose shoulders"). The most striking features in the anterior wall of the chest are the flattening in the region of the pectoralis and the peculiar folds of skin on the anterior edges of the axillæ.

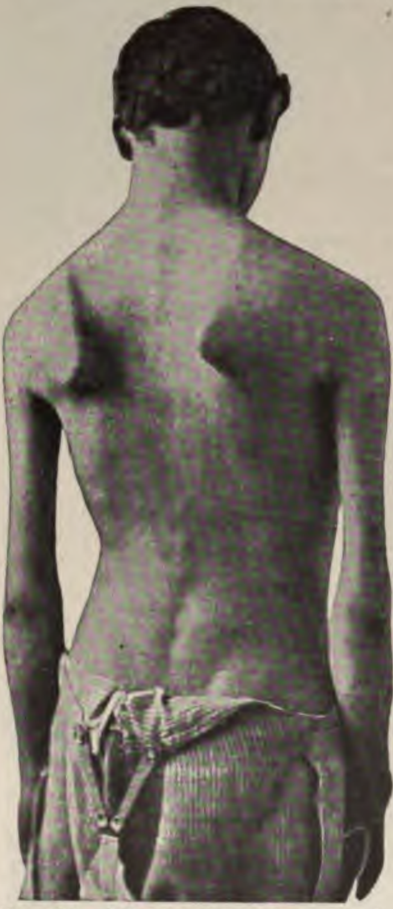

FIG. 183.—Muscular dystrophy. Prominence of the scapulæ when the arms are hanging down. (Erlangen Medical Clinic.)

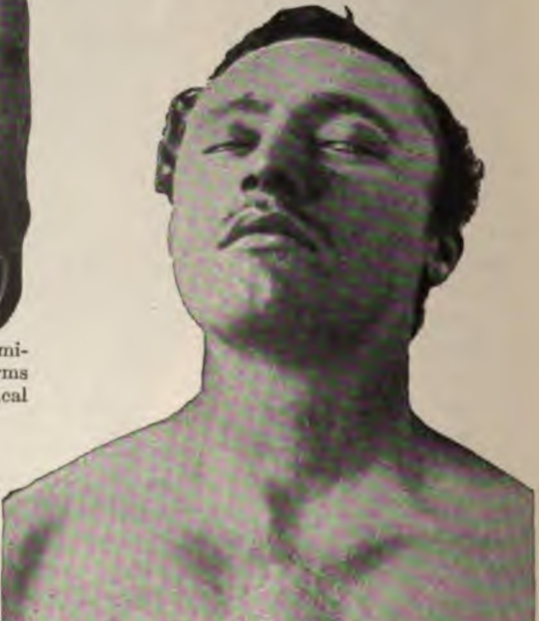

FIG. 184.—Juvenile muscular dystrophy. Insufficient closure of the eyes. Myopathic facies. (Personal observation.)

If the patient walks we can often recognize the disease at a distance at the first glance by the peculiar gait. The upper part of the body is carried backward (lordosis of the lumbar vertebræ), and the gait is waddling, since the pelvis can no longer be held properly by the weak glutæi. On stooping and rising, the same symptoms are seen as have previously been described (Fig. 178). As a rule, in the juvenile form the face is not involved, but we can sometimes detect slight disturbances of the facial muscles, especially defective closure of the lids and inability to whistle (Fig. 184). We once made the interesting observation that the adult brother of a young girl suffering from juvenile dystrophy showed these (congenital?) anomalies in the face from earliest childhood, without showing any further indications of dystrophy. Bulbar symptoms are as rare as in the other forms. It is of significance that the intercostal muscles and the diaphragm may finally atrophy, and the consequent respiratory disturbance may be the cause of death.

The course of juvenile dystrophy is very chronic, like that of all the other forms. The disease progresses very slowly and often seems almost to stand still for a long time. Many cases have been under observation for twenty or thirty years.

We may mention in conclusion that in some cases of dystrophy the trouble has been seen to begin at a more advanced age—from forty to fifty—but in other respects these cases do not differ from the general type, although we have noticed that, in such cases as begin relatively late in life, the muscles of the pelvis and thighs were chiefly affected, so that the patient could not stand or walk while the arms still performed their functions tolerably well (Fig. 185).

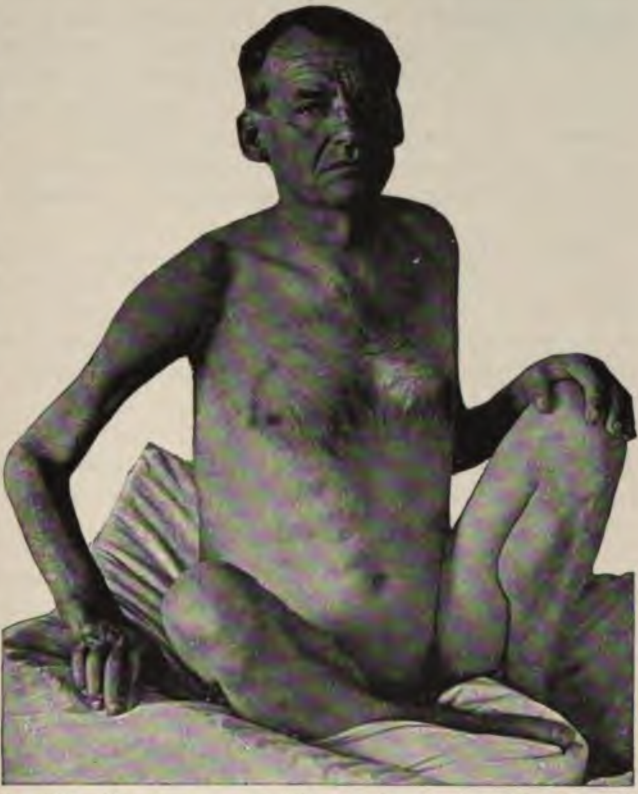

FIG. 185.—Progressive myopathic muscular atrophy of the pectorals, upper arms, and muscles of the pelvis and thigh. Ordinary position of the patient in sitting; he can neither stand nor walk. (Personal observation.)

Diagnosis of Myopathic Muscular Atrophy (Muscular Dystrophy).—Now that the type of myopathic muscular dystrophy has been established in all its details by numerous observations, the diagnosis is usually no longer difficult. The generic occurrence of the disease, its development usually in childhood or youth, the remarkable regularity in the selection of muscles affected, the absence of fibrillary contractions and also of electrical reaction of degeneration in the affected muscles, are the chief points upon which the diagnosis is to be based in the individual case. If we bear in mind these peculiarities, the distinction from the spinal and neurotic amyotrophies or from other similar types of disease (syringomyelia) is usually easy. Only a few cases, which clinically seem to present transition forms, remain for a time uncertain in their anatomical determination. We must, however, indicate one condition which may sometimes give rise to mistakes in diagnosis—namely, *congenital muscular defects*. In some persons we find that certain muscles or parts of muscles are absent from birth, and it is remarkable that it is generally the same muscles which are most often affected in muscular dystrophy, especially the pectoralis major, or at least its sternal portion, the serrati, the rhomboidei, the trapezii, etc. Such congenital muscular defects naturally present at the first glance a very similar picture to that of a patient with dystrophy; but we can usually decide correctly as to the condition by careful inquiry, since in case of congenital muscular defects the abnormalities have always been present, and they also show no noticeable increase in the course of years.

Treatment.—The treatment of myopathic muscular dystrophy is seldom successful. We may sometimes obtain a certain improvement by persistent treatment by electricity or massage; but at present any considerable alteration in the course of the disease is beyond the domain of possibility. [Persistent and systematic gymnastic exercises sometimes have a decidedly beneficial effect.—K.]

6. PRIMARY DEGENERATION OF THE PYRAMIDAL TRACTS

(Primary Lateral Sclerosis. Spastic Spinal Paralysis. Motor Tabes. Spasmodic Tabes Dorsalis)

While in all the forms of primary disease of the motor system thus far described the muscles are involved primarily or secondarily, and muscular atrophy therefore plays a leading part in the clinical picture of all the diseases described above, in rare cases the degeneration may be limited to the central neuron (*PyS* of Fig. 168, page 368) of the motor tract. In such cases a type of disease develops, in which indeed the disturbances are exclusively motor, but in which muscular atrophy is completely absent at least for a long time—a type of disease to which has been given the name of "*spastic spinal paralysis*" (Erb, Charcot). There is no doubt that such primary, isolated degeneration of the pyramidal tract may occur, without involving the peripheral motor neuron (cell of the anterior horn with its process, the peripheral motor nerve); but we must emphasize that the degeneration is not always limited to the lateral pyramidal tracts in the strict sense, but frequently affects the motor fibers of the antero-lateral tracts in their entirety. Moreover, primary degeneration of the pyramidal tracts is often found associated with degeneration of other neuron systems (lateral cerebellar tracts, posterior columns). Thus, the anatomical picture of a "combined tract disease" arises.

Type of Spastic Spinal Paralysis.—Three symptoms predominate in the picture of spastic spinal paralysis: motor paresis, hypertonicity of the muscles (muscular rigidity), and the increase of the tendon reflexes—the patellar reflex, and ankle clonus. The loss of motor power—we are speaking at present only of the spastic paralysis of the legs, which is by far the most frequent and the most clearly marked—is found in various degrees, from a simple weakness of movement to a complete and more or less extensive paralysis.

As a rule, only the flexors (shorteners) of the legs become paretic at first, while the extensors still retain their full power. But flexion of the leg is also altered in a peculiar way: with each attempt to draw the leg toward the trunk, a coincident marked contraction of the tibialis anticus muscle, which cannot be suppressed by the patient, and which can be recognized by the prominence of its tendon (so-called tibialis phenomenon), ensues. Sometimes (just as hemiplegics) the patients cannot extend the foot dorsally of their own accord, whereas if the entire leg is drawn up, a marked dorsal extension of the entire foot occurs. This phenomenon is explained by the reason that, in consequence of the degeneration of the lateral pyramidal tracts, the innervation of the muscles can only occur in groups ("synergically") and no longer in the single muscles. The pyramidal tract is probably principally a tract for the finer graded and isolated motor stimuli, while the other motor paths of the antero-lateral tracts serve more for the coarser movements executed by entire groups of muscles. If therefore a complete paralysis of all the muscles finally ensues, we can assume with certainty an extension of the degeneration beyond the limits of the lateral pyramidal tracts to all the antero-lateral motor tracts.

The characteristic "spastic" feature of the paralysis is in all probability due directly to the degeneration of the fibers of the lateral pyramidal tract. The fundamental clinical symptoms of "spastic paralysis" due to this degeneration are the hypertonicity of the muscles and the increase of the tendon reflexes. Both symptoms, indeed, are to be regarded as in part identical, for the rigidity of the muscles may also be of reflex origin. If the tendon reflexes be much increased, the reflex tension of the muscles comes on even upon the stretching and pulling of the tendons, which are excited by the weight of the limbs, or by any active or passive movements. The reflex muscular tension opposes any attempt at motion. If we try to flex the leg passively at the knee, or if we try to extend the foot dorsally, we find it almost impossible to do so. The more rapidly and suddenly we try to produce the motion, the more marked is the muscular resistance, which can scarcely be overcome. If, however, we go to work very slowly and cautiously, and avoid any sudden tension of the tendons, we can almost always flex the leg without special trouble. If we put the patient on the edge of the bed, the legs do not hang down laxly, but they usually fall at once into a vigorous extensor tetanus, since the weight of the leg puts the quadriceps into contraction by the tension on the ligamentum patellæ. A convulsive, reflex tremor of the whole leg often comes on at once, similar to ankle clonus. If we examine the patient while in the bath we find the spasms decidedly less, because in the water the influence of the weight of the limb is diminished. In addition to the reflex rigidity caused by external mechanical stimuli, a direct hypertonicity of the muscles (an increase of the continuous mean muscular tonus caused by a loss of the depressor fibers which normally affect it) is frequently present. In many cases the muscles appear continually rigid and tense to the touch, the legs are in a condition of continual extensor tetanus and adductor contracture. If we attempt to elevate one leg passively or abduct it from the body, the other leg and the entire body soon follow. In the dorsal flexors of the toes, especially of the great toe, this continued hypertonicity is, as a rule, very evident. The characteristic phenomenon found in all spastic paralyses of the legs

—viz., that the cutaneous reflex of the great toe produced by stroking the sole of the foot [or irritating the tibial region] occurs as a dorsal extension (so-called Babinski [and Oppenheim] reflexes)—probably depends on the above circumstances.

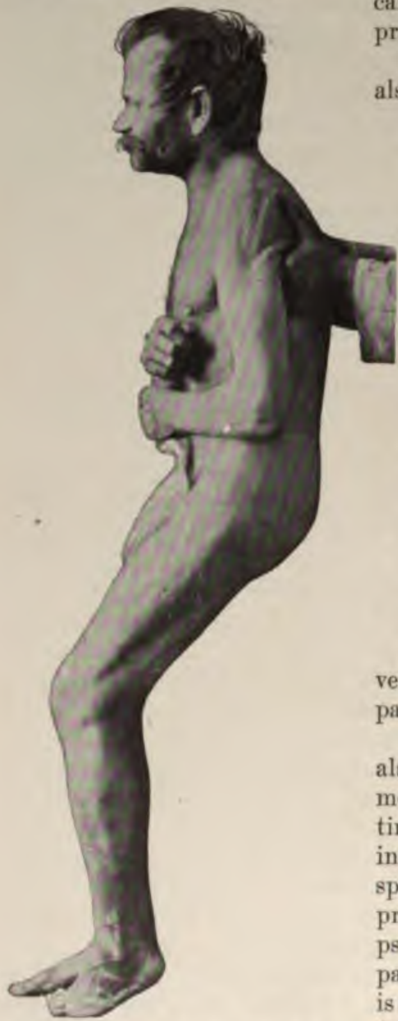

FIG. 186.—Spastic spinal paralysis. Enormous muscular rigidity of the trunk, arms, and legs. When supported from behind, the patient can stand as stiff as a post. (Erlangen Medical Clinic.)

Active motion, as is easily explained, must also be impaired from the inhibitory action of the tonic and reflex spasms. The degree of disturbance of motion is thus increased, and the paresis often seems greater than is really the case. The influence of the muscular tension is especially manifest in the gait of the patient. As long as walking is still possible, we notice very plainly that it is rendered difficult, not only by the muscular paresis, but also by the stiffness of the legs. The patient walks with short and difficult steps, the legs are scarcely flexed at the knee, and the feet are scarcely raised at all. The feet "stick to the floor" and are slowly slid forward, and there is a marked tendency to walk on the toes, from the contraction in the muscles of the calves. The weight of the body alone presses the feet downward. We term this very characteristic form of gait the spastic-paretic gait.

The increase in the tendon reflexes may also exist without the presence of any special motor paresis of the muscles at the same time, but since the motion is not a little influenced in such a case by the constant spasms, a disturbance of motility may be produced, which we have called "spastic pseudo-paralysis," or, more properly, pseudo-paresis. In these cases the muscular strength is almost normal, and the patient can walk for quite a long time. Nevertheless, all his movements are stiff and difficult, and the gait shows all the peculiarities of the pure spastic gait. The steps are not very short and they follow one another quite rapidly, but the legs remain perfectly stiff and are scarcely raised from the ground, and the patient walks almost entirely on his toes. In the house the patient walks with a noisy shuffle, and in soft sand we can see the furrows drawn by the feet as they slide along the ground. Since the Achilles tendon is made tense at each step, and since a reflex contraction of the muscles of the calf responds to this tension, each step is often followed by a short lifting of the whole body ("teetering" spastic gait).

Although the symptoms mentioned are the characteristic positive features of "spastic spinal paralysis," yet, in the original sense of the word, the affection is also characterized by the fact that certain other spinal symptoms, especially disturbances of sensibility, disturbances in micturition and defecation, muscular atrophy, and other trophic symptoms, are entirely absent. Only when such was the case have Erb and Charcot claimed that a special anatomical cause must lie at the bottom of the peculiar group of symptoms, which they suspected to be a primary disease of the lateral columns—that is, of the pyramidal tracts particularly. This suspicion was entirely warranted, since the picture of pure spastic spinal paralysis could be explained by no other anatomical theory. A degeneration in the lateral columns explains the motor paresis, explains the increase of the tendon reflexes by the loss of reflex inhibitory influences, and the increased tonic tension of the muscles. Actual evidence of the occurrence of primary systemic degeneration in the lateral columns could, of course, be furnished only by anatomical observation. This has now actually taken place, so that at present there can no longer be any doubt of the occurrence of a "spastic spinal paralysis" in the original sense of Charcot and Erb. If we observe the same morbid picture not only in primary degeneration of the pyramidal tract, but also in other cases, it is due to the fact that, under certain circumstances, other diseases may, at least for a time, involve the lateral columns almost exclusively, and especially the pyramidal tracts.

So far as our present knowledge extends, the affection comes on in two somewhat distinct clinical forms. The first may be brought into the closest relation with amyotrophic lateral sclerosis. As we have seen above (page 371), we often find in typical amyotrophic lateral sclerosis a pure spastic paresis in the legs without any trace of muscular atrophy, while in the arms and the bulbar region there is more or less marked muscular atrophy besides the spastic symptoms. There are also cases of disease—they occur usually in somewhat advanced life and sporadically; that is, not generically as

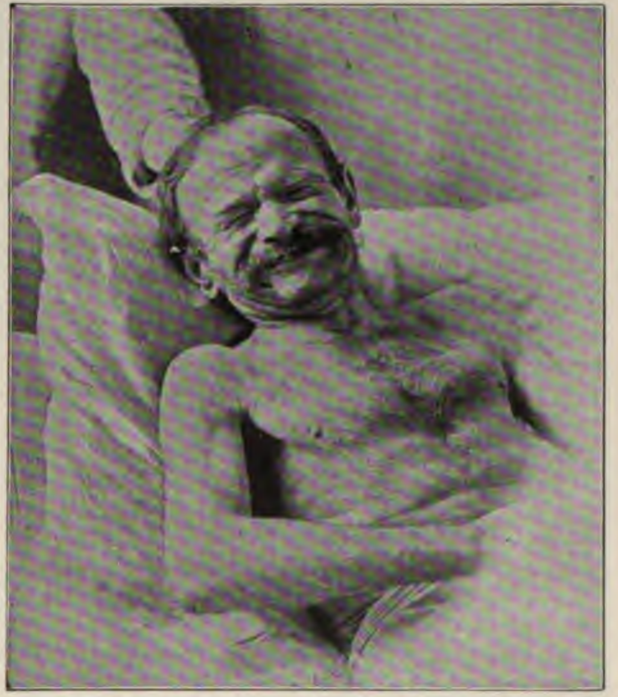

FIG. 187.—Spastic spinal paralysis. Impulsive laughing. The same patient as Fig. 186. Symptoms of amyotrophic lateral sclerosis came on later. (Erlangen Medical Clinic.)

a family disease—where spastic rigidity and paresis develop not only in the legs, but also in the trunk, the arms, and the face, without a trace of muscular atrophy. In the course of a few years the patient finally becomes almost completely stiff (Fig. 186). The stiffness is due solely to the muscular rigidity, and only on the further progress of the disease does the muscular rigidity eventually go on to complete paralysis. The arms also show pronounced hypertonicity on passive movement. Impulsive laughing and crying also occur, just as in amyotrophic lateral sclerosis (Fig. 187). If we have the rare opportunity to make an autopsy on such a case, we find a marked systemic degeneration in both lateral columns, chiefly in the region of the pyramidal tract, *PyS*, but, especially if the case had progressed to a condition of true paralysis, involving other bundles of fibers in the antero-lateral columns as well. The degeneration of the pyramidal tract, *PyS*, may be traced upward through the pyramids, the pons, and the crus to the internal capsule. The ganglion cells in the anterior horns of the spinal cord are entirely normal or they show only insignificant changes. The close relationship between this form of "pure lateral sclerosis" and amyotrophic lateral sclerosis is shown by the fact that the one disease may develop into the other. After the typical affection of lateral sclerosis has continued in its pure form for some years, atrophy may sometimes finally develop in the small muscles of the hand, the tongue, etc.—in short, there is clinical evidence that the peripheral motor neuron has begun to degenerate; but the atrophy, as a rule, remains comparatively slight, although different degrees of transition forms probably occur.

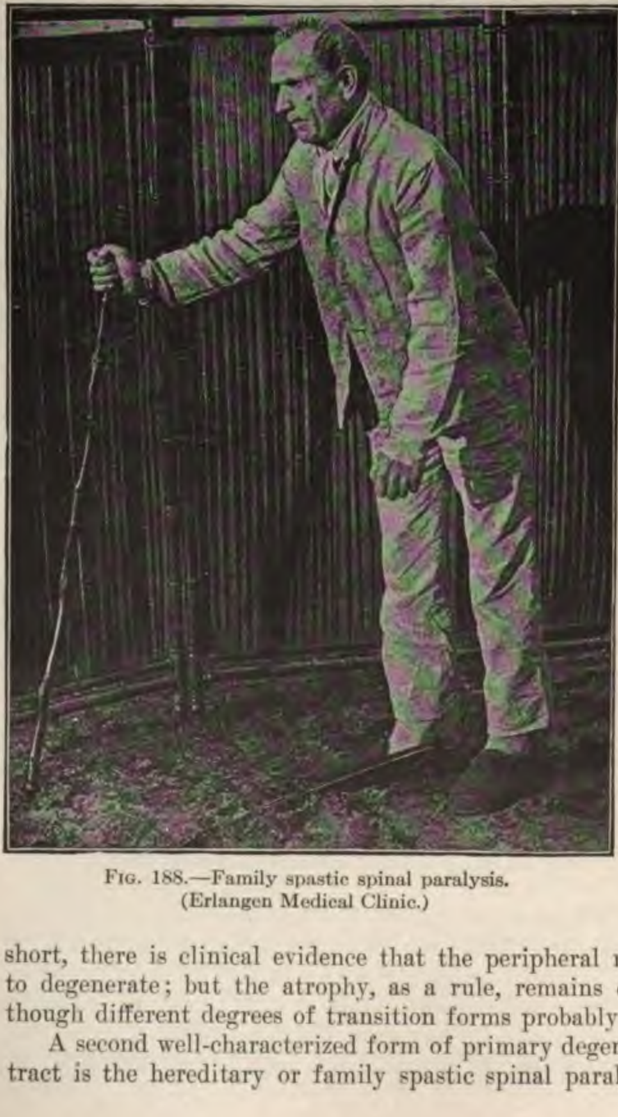

FIG. 188.—Family spastic spinal paralysis. (Erlangen Medical Clinic.)

A second well-characterized form of primary degeneration of the pyramidal tract is the hereditary or family spastic spinal paralysis which has been described by me. This shows itself in several members of the same family, especially in brothers and sisters; in this form there very gradually develops, in the course of years and years, a constantly increasing rigidity of the legs with increased tendon reflexes and all the motor disturbances dependent thereon. The gait is pronouncedly spastic, the feet cling to the floor, the legs are pressed firmly together from the contracture of the adductors, and on walking, one foot can pass the other only with difficulty. The patient treads only on the anterior part of the foot; the whole body is projected stiffly forward, so that the patient has to hold a cane in front of himself to keep from falling forward (Fig. 188). For a long time the purely spastic symptoms outweigh the paretic (spastic pseudo-paralysis), and only very gradually does the spastic gait become spastic-paretic, and an actual loss of muscular strength ensue. The general condition, the sensibility, the bladder and the muscular nutrition may remain unaffected for a long time or until the end. The disease usually begins somewhere between twenty and thirty, but some cases apparently occur in children also. There can hardly be any doubt that the disease is due to a congenital morbid predisposition of the pyramidal tract, and that thus it is closely connected, ætiologically, with the other forms of hereditary systemic disease, especially with hereditary muscular atrophy; but the anatomical examination of the spinal cord, which has been possible thus far in only a few cases, has shown disease in the lateral cerebellar tract (*KlS*) and the columns of Goll, besides degeneration in the pyramidal lateral tract (*PyS*)—that is, in the territory of the neurons of the lumbar spinal ganglia. Anatomically, therefore, hereditary spastic spinal paralysis must also be termed a combined systemic disease.

Besides the two forms of primary degeneration of the pyramidal lateral tracts (*PyS*) already mentioned, which in our opinion are perfectly well established clinically and anatomically, there are in reality several other forms of disease whose clinical symptoms are in the main those of spastic spinal paralysis, while the anatomical changes consist of a primary, more or less strictly systemic disease of the lateral columns alone, or of the lateral and posterior columns. In the latter case ataxia, bladder disturbances, slight disturbances of sensation, etc., may at times be added to the spastic symptoms; but since we have very little definite knowledge of these rare forms of disease, we cannot at present give any suitable description of them. Many of these diseases seem to be exogenous in origin and to be especially connected with previous syphilis. Other cases are related ætiologically to severe general constitutional diseases, especially pernicious anæmia (*q. v.*). We may also mention that a pure type of spastic spinal paralysis may be produced by chronic poisoning with lathyrus seeds (lathyrus sativus and lathyrus cicera, the so-called vetches, which are used to make bread in southern countries). Systemic degenerations of the spinal cord also occur in pellagra [see Appendix], which is common in northern Italy from the continued use of diseased or fermented maize. This sometimes causes especially marked spastic symptoms, besides, of course, many other spinal and mental symptoms (Tuczek).

[Gowers has given the name of *ataxic paraplegia* to a class of cases of combined degeneration of the lateral and posterior columns, which are by no means rare. The disease comes on gradually in middle life, usually without any definite cause. Men are much more frequently affected than women. The early symptoms resemble those of spastic paraplegia, with the addition of ataxic unsteadiness, which may be at first the most prominent symptom. There are very rarely any lightning pains, but there is at times a dull pain in the legs or the sacrum. There is rarely any impairment of sensation. The tendon reflexes are usually much exaggerated. Sexual power may be lost early, and the bladder and rectum are often affected. Reflex immobility of the pupils and optic atrophy are rare, but nystagmus is occasionally noticed. The arms are usually involved later. Mental symptoms are rare. The disease is slowly progressive, but it rarely proves fatal, death being usually due to some intercurrent disease. The extent of the degeneration in the cord varies, but it is never so well marked in the posterior columns as it is in true tabes. Putnam has described a type of degeneration occurring also in adult life, but much more frequent in women. In his cases the trouble seemed to be not infrequently associated with profound anæmia or other forms of malnutrition. Paræsthesia is a very prominent symptom of the earlier stages of the disease, and it may come on suddenly or rapidly increase. Its appearance in an anæmic person of middle age is therefore suggestive of the onset of this form of disease. Disturbances of micturition are also very common in the early stages. Incoördination and muscular weakness develop later, and the weakness may increase to complete paraplegia. The reflexes are usually exaggerated, but they may be absent. The disease is apt to terminate fatally within four or five years. The pathological changes are of a degenerative nature, and are most marked in the posterior and lateral columns, but they are quite diffuse, so that this type of disease is more closely allied to diffuse chronic myelitis than to a systemic degeneration. In some cases systematic gymnastic exercises, by Frenkel's method, prove of decided benefit. The treatment otherwise is the same as that of chronic myelitis.—K.]

Diagnosis.—Since the symptom-complex of spastic spinal paralysis must always arise if the pyramidal tracts are affected by any disease in any part of the two lateral columns of the spinal cord or even higher up, it is easily seen that it may often be very difficult or even quite impossible to decide in a given case whether we have to do with a primary systemic degeneration or with some other circumscribed disease of the pyramidal tracts. The chief weight in diagnosis should always be laid on the general development of the morbid condition, the ætiological conditions (several in a family affected), and the strict limitation of the symptomatic picture (absence of any sensory or bladder disturbances, etc.).

The morbid conditions chiefly to be considered in differential diagnosis are the following: (1) *Multiple sclerosis.* As previously stated (page 330), this may for some time resemble very closely the type of pure spastic spinal paralysis. (2) *Syphilitic disease of the upper dorsal cord,* the so-called *syphilitic spastic spinal paralysis.* We will consider this important disease more fully in a subsequent chapter on syphilis of the spinal cord. (3) *Chronic hydrocephalus* (*vide infra,* the section on diseases of the brain). (4) *Subacute and chronic myelitis* following other acute diseases (typhoid fever, influenza, etc.). This can, as a rule, be easily distinguished from genuine spastic spinal paralysis by the mode of origin, and also by the clinical symptoms. (5) Slight *compression of the spinal cord,* from vertebral caries, tumors, traumatic affections of the vertebræ, etc., may for some time give rise to typical symptoms of spastic spinal paralysis, but with careful attention and longer observation there will only rarely be any mistake.

Prognosis and Treatment.—The prognosis of genuine spastic spinal paralysis is naturally as unfavorable as that of any other systemic disease. The treatment can therefore be merely symptomatic. The spastic symptoms, which are often very troublesome, are most readily alleviated by the use of long-continued warm baths. In other respects the treatment follows the rules that obtain in all chronic affections of the spinal cord.

APPENDIX

CONGENITAL SPASTIC PARAPLEGIA IN CHILDREN

(*Spastic Cerebral Paralysis. Infantile Spastic Diplegia. Little's Disease*)

IN this place we would also speak briefly of a morbid condition, first described by J. Heine and the English physician Little, which is not very rarely observed in children. The symptoms in these cases are probably usually congenital, or at least they arise during birth (*vide infra*), but from reasons that are easy to understand they are not noticed until later. When the children come under medical observation they are usually from three to six years old, or even older. The most striking symptom is that the child cannot walk at all, or he walks only with difficulty and with assistance. If we examine the legs carefully we usually find that they are quite stiff, and have assumed a characteristic change of position. The thighs are usually somewhat flexed and firmly adducted, and also rotated inward. The knees are also slightly bent, while the feet are often in a position of equinus or equino-varus (Fig. 189). We suspect that this peculiar condition of the legs which is found, as a rule, in infantile spastic paresis, but is never found in this form in the spastic paresis of adults, is due to a retardation of the growth of the flexors, which are principally paralyzed.

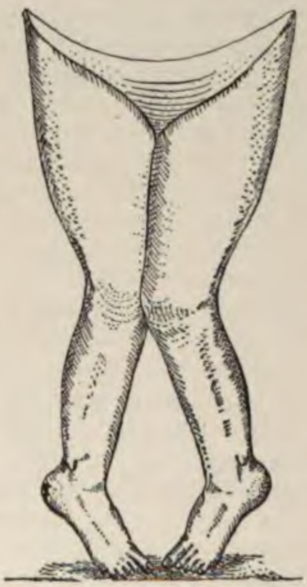

FIG. 189.—Position of the legs in congenital spastic cerebral paralysis. (Sketch from personal observation.)

From the contracture of the thighs in adduction it readily happens that the legs cross each other when the child lies down, or especially when he tries to walk. Passive movements of the legs can be performed only by overcoming more or less muscular resistance. The tendon reflexes, especially the patellar reflex, are much increased, and there is often a persistent ankle clonus. The sensibility and micturition are normal. The arms, as a rule, are readily moved, but slighter spastic symptoms may also occur in them. Many of these children are mentally perfectly normal, learn to talk and write well, etc.; but in other cases cerebral disturbances are also plainly manifested, feeble intelligence, defective speech, epileptic attacks, strabismus, etc. As the child's physical development proceeds the symptoms often improve, but in general the condition is stationary and not progressive.

Our knowledge is still very defective as to the causes of these congenital spastic paraplegias and the anatomical changes which underlie them. First of all we must make a fundamental division of the cases into two groups. In the one we have to do with children who are born with great difficulty and with artificial aid (forceps, etc.). In this group we can probably assume injuries of the brain during parturition from hemorrhage, etc., which result in permanent disturbances of function. In the other group, where the history affords no such evidence (except that often the children are born prematurely), we usually have to assume some congenital defect (so-called porencephaly in the cerebrum, congenital aplasia of the motor centers or of the whole pyramidal tract). This congenital form of spastic paraplegia may also occur generically, but it is to be distinguished from the hereditary spastic paralysis above described, which develops slowly and only in later life, and is progressive. However, some observations appear to us to favor the belief that there is a "true spastic spinal paralysis," which may already develop during childhood.

In the way of treatment, we may expect most from regular gymnastic exercises. Surgical interference (tenotomy, transplantation of tendons) and orthopedic appliances may sometimes considerably improve the child's ability to walk.

CHAPTER X

ACUTE AND CHRONIC POLIOMYELITIS

1. SPINAL PARALYSIS OF CHILDREN

(Acute Poliomyelitis in Children)

Ætiology and Pathological Anatomy.—In children there occurs quite frequently a definite and well-characterized form of paralysis, for the first accurate knowledge of which we must thank Jacob von Heine in 1840. Although Heine subsequently, in 1860, expressed the opinion that a disease of the spinal cord formed the basis of the paralysis, the first actual confirmation of this opinion was furnished by Prévost and Vulpian, Charcot and Joffroy, and others, so that at present we are justified in exchanging the old term "essential paralysis of children" for the name of "spinal paralysis of children." The opinion I have held for a long time that ætiologically entirely similar inflammatory processes can occur in other parts of the nervous system (brain, pons, etc.), has been recently confirmed, especially by Scandinavian investigators. Wickmann has proposed to group all these different localization types of the same disease together under the name of "Heine-Medin's disease." [In the recent epidemic in New York the spinal type was found to be the predominating form, and all other forms, the ascending type, the bulbar, pontine, cerebral, ataxic, polyneuritic, meningeal, and abortive types represented rare varieties. For this reason the committee who investigated the epidemic felt that there was not sufficient reason to adopt the term Heine-

Medin's disease in place of the old name epidemic poliomyelitis. No case of the cerebral cortical type was observed in the New York epidemic.—K.]

As the name indicates, the affection occurs chiefly, if not exclusively (*vide infra*), in children, and is most frequent in the earlier years, somewhere between one and four. [In the recent epidemics in New York and Massachusetts from seventy to eighty per cent of the cases were under five years of age.—K.] An exciting cause, such as taking cold, has hardly ever been made out. The children are almost always perfectly healthy[1] previously, and come of healthy families without any neuropathic predisposition. The whole course of the disease makes the hypothesis very probable that we have to do with an acute infectious disease—with an infectious process, which often causes first a mild or severe general infection of the body, and then is localized chiefly in one or several circumscribed portions of the spinal cord. It also, perhaps, bears some relation to the infectious nature of the disease that most of the cases occur in the summer and autumn. The fact should also be mentioned that small and even extensive epidemics of spinal paralysis of children have been repeatedly observed. We have ourselves seen three children fall ill in a little village in a few days with acute poliomyelitis. Very extensive epidemics of acute poliomyelitis have recently been observed in Scandinavia and Austria. Here the mode of extension of the disease appeared to point to a possibility of its direct transmission and contagiousness.

[There seems to have been a very great increase in the disease in recent years. In the reported epidemics since 1880 less than 1,000 cases were reported for the period from 1880 to 1905 and over 8,000 cases since that time. The most extensive epidemics known have occurred in the United States in the years 1907–10. It is believed that fully 20,000 cases occurred in the United States during that time. Several hundred cases occurred in Minnesota, over 600 in Nebraska, nearly 1,300 in Massachusetts (Lovett), and at least 2,500 in New York, especially in the vicinity of New York City. The New York epidemic seems to have spread along the ordinary routes of travel, and was possibly the source of the epidemic in Massachusetts. In 1907 the epidemic reached its height in New York in September, having begun in June, and it seemed a trifle later in Massachusetts. The investigations in New York and Massachusetts indicate direct contagion in a certain number of cases, with a period of incubation of from one to fourteen days. The infection may possibly be transmitted by a third person, and dwelling houses may become infected.—K.]

With reference to its anatomy, the disease may be defined as an acute inflammation which affects chiefly a definite portion of the anterior gray matter of the spinal cord, usually attacking only the anterior gray horn of one side; yet it does not always limit itself strictly to this, but it generally involves the white matter in the vicinity somewhat, although, of course, to a small extent. According to the recent careful investigations of J. Wickmann, Lövegren, and others, we are dealing with a true acute hemorrhagic inflammation, which is generally much more extensive anatomically than we would assume from the clinical symptoms. Indeed, a careful examination of the entire nervous system usually reveals that in addition to the main poliomyelitic focus there are very frequently smaller inflammatory foci in other parts of the spinal cord, the medulla, and even in the cortex of the brain. The meninges are only secondarily affected by the inflammation. All investigations of the bacterial cause of the disease have so far been in vain. The lesion which is most frequently found in old cases consists of a considerable atrophy of one anterior horn, which is cicatrized, shriveled and changed to a dense sclerosed tissue, often pierced by dilated and thickened vessels, and containing scarcely a single normal ganglion cell. If the paralysis affects one arm, the corresponding anterior horn in the cervical enlargement is atrophied (see Fig. 190); if the leg be paralyzed, the process is seated in the lumbar enlargement. In bilateral paralysis we must think of an affection of both anterior horns at the corresponding level of the cord. The entire lesion is therefore to be classed as a disseminated myelitis. Nevertheless, the predominating involvement of the gray anterior columns, which is already evident from the clinical symptoms, is worthy of note. For the present, we are unable to offer an explanation of this fact.

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[1] The paralyses arising after acute diseases—such as measles, scarlet fever, smallpox, etc.—are perhaps partly of spinal origin, but, without further inquiry, they cannot be identified with the idiopathic spinal paralysis of children.

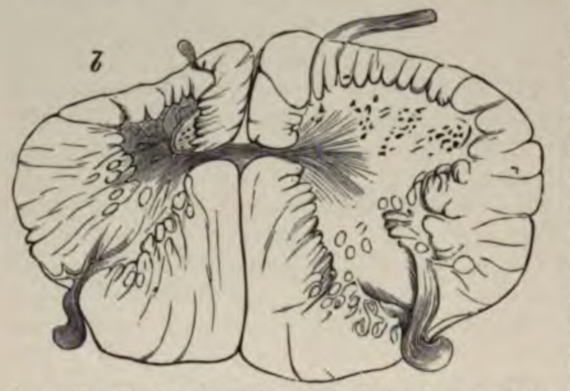

Fig. 190.—Section through the cervical enlargement in anterior poliomyelitis; the left anterior column is very much contracted and is without ganglion cells. (From Charcot and Joffroy.)

This inflammation of the anterior horn, poliomyelitis, is to be regarded as the primary focus of disease, dependent upon the local action of the excitants of inflammation. From this point, as in every severe lesion of the motor ganglion cells there situated, there develops a secondary degeneration, which, extending to the periphery, affects the corresponding anterior roots, and later their appropriate motor nerves and the muscles supplied by them. In the paralyzed muscles and nerves we accordingly find a high degree of degeneration and atrophy, such as we have learned to recognize in severe peripheral paralyses.

[Strauss, in New York, has confirmed many of Wickmann's investigations. The interstitial changes in the cord are of fundamental importance, and the ganglion cells are affected only secondarily. The interstitial process is dependent upon its relation to the vessels for its character and localization. The lesions are most marked in the anterior horns, but the whole gray matter of the cord is apt to be affected so that the word "anterior" may be dropped. Infiltration of the pia is also an essential element. The medulla, pons, and basal ganglia are always involved in fatal cases, but the ganglion cells in these regions generally escape serious morphological alteration. The œdema of both white and gray matter is an important factor in producing the paralysis and explains the transitory character of some of the symptoms. The spinal ganglia may also be infected, and the infection may involve any part of the nervous system. The central artery of the cord does not have a predominating part in the origin of the disease, and there is no evidence of thrombosis. Flexner and Lewis have proved conclusively that the disease can be transmitted to monkeys by inoculation with the spinal cord of human beings who have died from poliomyelitis, and can again be transmitted by reinoculation from monkey to monkey. The virus resides in the brain as well as in the cord, but the cerebro-spinal fluid does not contain it. This virus is not bacterial, but may be of protozoal nature. Inoculations may be subdural, intravenous, into the nerve sheaths, or intraperitoneal. The subdural inoculation is probably the surest. The incubation period after inoculation was from four to thirty-three days. The lesions from poliomyelitis and the routes of infection in monkeys have suggested some relation to rabies, although Lovett could find no connection between the two diseases in the Massachusetts epidemic of 1909. Flexner has suggested that the infection may take place through the nasal passages.—K.]

Clinical History.—The disease almost always begins suddenly. A child who was previously perfectly well and lively is all at once attacked with severe fever, often reaching 105° or 106° F. (40° to 41° C.), and quite severe general symptoms are also associated with the fever even from the beginning. The child complains of headache, and sometimes of pain in the loins and in the limbs, and is decidedly stupid and somnolent. Very often still more marked cerebral symptoms develop—complete loss of consciousness, single twitchings in the face or the extremities, or general convulsions. The eclamptic attacks, turning of the eyes, and clonic contractions in the face and extremities, sometimes appear even at the beginning of the disease. On the part of other organs we see most frequently gastrointestinal symptoms, especially vomiting, more rarely diarrhea. [In a considerable number of cases in the New York epidemic, coryza, bronchitis, and sore throat were among the early symptoms, pointing to Flexner's hypothesis that the upper respiratory tract is the seat of invasion. This was not the case in the Massachusetts epidemic, where gastrointestinal symptoms played the leading part. Restlessness, headache, delirium, convulsions, and apathy are very common. Pain and tenderness in the legs and spine are extremely common, and there is occasionally rigidity of the neck and Kernig's sign, so that cases may be mistaken for meningitis. Lumbar puncture will determine the diagnosis. General symptoms of infection are sometimes as prominent as paralysis.—K.] All the initial symptoms, whose intensity varies very much in the different cases, sometimes last only a very short time—a day or two—although they often continue for a week or two. Indeed, we often know cases in which, as the mothers have assured us, the children are said to have "lain in spasms," almost uninterruptedly, even for four or five weeks before the beginning of the paralysis—that is, before it became noticeable. On the other hand, however, it may happen that the initial symptoms, especially the severe cerebral symptoms, are entirely absent or greatly modified. Occasionally a mild sore throat is found at the onset.

After the initial period of the disease just described has passed away, the parents usually notice that the child is attacked by a more or less extensive paralysis. If its development can be followed closely, we always find that it spreads rapidly, often in single spurts which quickly follow one another, so that it usually reaches a considerable extent in a short time. Either both legs, or the legs and one arm, or all the extremities, and even the muscles of the trunk, are affected. In some cases one oculomotor or facial nerve has been observed to be affected at the beginning. The paralysis scarcely ever remains permanently as extensively distributed as at first; it diminishes rapidly, and soon is limited to a definite muscular region, which remains permanently paralyzed. In some cases the paralysis may entirely disappear, but, as a rule, a complete paralysis is left in one extremity, or at least in a portion of it; most frequently in one leg, especially in the peroneal muscles, in other cases in the muscles of the thigh; somewhat more rarely in the arm, chiefly in the deltoid, in the upper-arm muscles, and much more rarely in muscles of the lower arm and of the hand; sometimes in both legs, or, rarely, in spinal paralysis, in one arm and leg on the same side or on opposite sides. [In the New York and Massachusetts epidemics both legs were very often, and all four limbs quite frequently, affected.—K.] It is important in all severe cases not to neglect a careful examination of the muscles of the trunk. Poliomyelitic foci in the dorsal segment of the spinal cord often produce very marked paralysis of the muscles of the back, of respiration, and of the abdomen. In this way marked scoliosis, etc., frequently arise. Meantime the child's general health has been completely restored. He is well and vigorous, has an excellent appetite, never shows any permanent cerebral disturbance—only the painless, flaccid paralysis, the inability to use the affected extremity, is left behind. In the following weeks and months a further improvement in the power of motion often becomes noticeable, and may go on to complete recovery, but, as a rule, permanent and more or less complete paralysis of certain muscles remains. If a careful examination of the functions of the muscles is now conducted, we can determine fairly accurately the location and extent of the poliomyelitic foci by referring to the data which have thus far been collected on the location of the spinal centers of the individual muscles. Accurate examinations of this sort are of interest also because they furnish us valuable information regarding the relative positions of the individual spinal centers of the muscles.

In regard to the more intimate peculiarities of this remaining paralysis, it may invariably be characterized as a flaccid atrophic paralysis. A marked atrophy of the paralyzed muscles shows itself a few weeks after the beginning of the paralysis. This atrophy gradually spreads, and it may finally attain the highest degree. The atrophy is often, but not always, partly concealed by a more abundant development of fat tissue. The changes in the electrical excitability of the paralyzed nerves and muscles come on still more rapidly than the visible atrophy. Since we have to do with a complete degeneration of the nerves, as follows from the anatomical basis of the disease, a pronounced electrical reaction of degeneration must necessarily develop in the affected parts. Duchenne found that usually the faradic excitability of the nerves and muscles is completely lost after a week or two. On galvanic examination, we can at first detect an increase of excitability in the muscles with a predominance of slow anodic closure contractions (*AnSZ*), while later, after two or three months, the galvanic excitability also sinks very considerably; but the muscular contractions preserve their qualitative peculiarities characteristic of reaction of degeneration (see page 218). Very often the whole affected extremity remains backward in its growth, so that later the bones may show a shortening of several centimeters (Fig. 191). The parallelism between the muscular atrophy and the stunted growth is not, however, present in all cases, as Volkmann has previously stated.

Passive motion of the paralyzed extremity is at first, on account of the absence or diminution of the muscular tonus, perfectly free, and so remains except for the contractures that finally set in (*vide infra*). Many joints are so flaccid that we can actually make flapping movements with the paralyzed limbs and give them the most extraordinary positions. The tendon reflexes are invariably completely absent in the paralyzed extremities, and so almost always are the cutaneous reflexes—a condition which is to be referred to the interruption of the reflex arc in the anterior horns, which may sometimes be of diagnostic significance. The skin often shows certain trophic disturbances (atrophy, anomalies in the growth of the hair and in the secretion of sweat); it almost always feels cool and has a cyanotic appearance. Its sensibility, however, is completely retained in all cases. According to our observation, a posterior poliomyelitis (inflammatory foci in the gray posterior horns) apparently also occurs, but only in isolated cases. Disturbances of pain and temperature sense are due to this lesion (*vide* page 219). Micturition is sometimes a little disturbed at the beginning of the disease, but in most cases this disturbance completely disappears later.

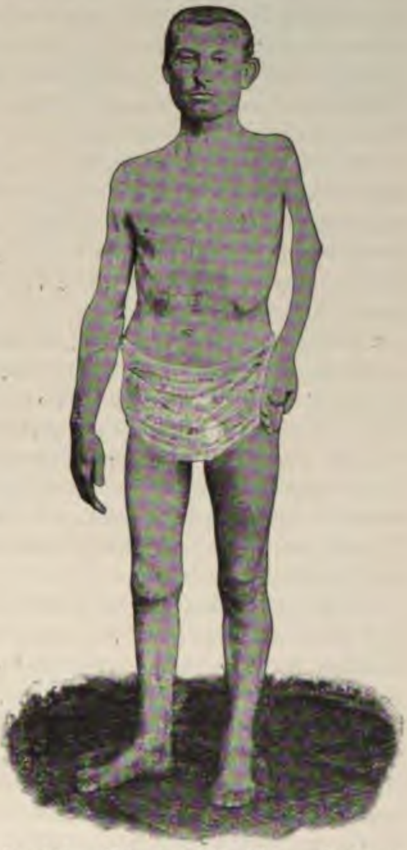

FIG. 191.—Old spinal infantile paralysis, acquired at the age of six. (Personal observation.)

After the paralysis has existed for a time, certain secondary contractures almost always develop in the paralyzed parts, some of which are of a very characteristic type. In the legs especially, the "paralytic clubfoot" (*talipes varo-equinus*) is a condition long known. It is due to the fact that, from the paralysis of the peronei muscles and of the tibialis anticus, the point of the foot constantly droops, and that a contracture is gradually developed in the antagonistic unparalyzed muscles of the calf, whose points of insertion are permanently approximated. In paralysis of the muscles of the calf there arises, on the other hand, a moderate degree of calcaneus from the contracture of the antagonists. In the arms and in the vertebral column, in paralyses of the spinal muscles, the most manifold and sometimes very considerable contractures and deformities may also arise, which are in the main always to be referred to the contracture of unparalyzed antagonists and to external mechanical conditions, such as weight and pressure.

In conclusion, if we simply compare the type of disease sketched with its anatomical cause, the general agreement of the two may at once be seen. The affection of the anterior gray cornua must have as a result a paralysis with a subsequent atrophy and reaction of degeneration, in which the reflexes must be lost by the destruction of the reflex arc, but the sensibility must remain perfectly normal from the persistence of the sensory conduction (the posterior columns and the posterior gray cornua), and the vesical functions must also remain normal. The subsequent paralysis is the result of the permanent destruction which the morbid process, in itself completely ended, has caused in the spinal cord.

We must also mention the peculiar fact which has been observed in some cases that progressive muscular atrophy may come on in later life in persons who long before have had spinal infantile paralysis. The cells of the anterior horns once injured by the acute disease seem never to have become equal to the functional demands made upon them, and therefore they succumb to a premature atrophy.

Diagnosis.—The diagnosis of the spinal paralysis of children is almost always easy to make correctly, if we hold strictly to the definition and peculiarities of the disease, and do not reckon as spinal paralysis every paralysis appearing in a child. We should consider chiefly the acute beginning, the subsequent flaccid paralysis with atrophy and reaction of degeneration, with the loss of the reflexes, but with retained sensibility. If we observe these features, we are sufficiently protected against confusion with cerebral diseases and other diseases such as spondylitis, hereditary muscular atrophy, or spastic cerebral and spinal paralysis.

It is quite possible that certain relations exist between acute poliomyelitis and acute polyneuritis. But we would constantly emphasize one principal distinction between polyneuritis and genuine, circumscribed acute poliomyelitis —polyneuritis has a general, hematogenous, toxic, or toxic-infectious origin, while acute poliomyelitis seems to be a true local inflammation dependent upon the local presence of the (organized?) excitants of inflammation. In the clinical differential diagnosis we should consider chiefly the existence of any initial pains and any initial sensitiveness of the peripheral nerves and muscles. [Pain and tenderness are, however, common in poliomyelitis, although usually to a less degree. The tenderness also is apt not to involve the nerves.—K.] The distribution of the paralysis in the different groups of muscles is almost invariably of a pronounced bilateral symmetrical type in polyneuritis, whereas the circumscribed inflammatory poliomyelitic process generally causes unilateral and asymmetrical palsies [*vide supra*]. The further course of the disease may also be decisive, since the neuritic paralyses afford a much better prognosis than the paralyses of poliomyelitis. The differential diagnosis between spinal and cerebral infantile paralysis will be discussed later on. [As fœtal poliomyelitis seems possible, there may, in rare cases, be some confusion with myotonia congenita (*q. v.*). In most cases the history of a definite onset with fever, etc., will make the diagnosis easy.—K.]

Prognosis.—It is not impossible, but also not yet proved, that many of the cases where children die speedily with convulsions are to be regarded as the initial stage of acute poliomyelitis. If, however, the first stage of the disease be past, the prognosis as regards life is entirely favorable, since the rest of the child's physical development is no further affected in any way. The prognosis as regards the complete restoration of the disturbance of function is, however, much more unfavorable. Although we should not forget that the paralysis is at first much more extensive than it is later, nevertheless, this lessening of the paralytic symptoms occurs only in the first weeks or months. A limb which has not recovered in six to nine months usually remains paralyzed for life. Nevertheless, this should not restrain us from persevering in treatment, at least in the first years, since sometimes a very noticeable improvement in the functions of the paralyzed parts is thus procured. [In the New York epidemic of 1907, which was on the whole of a mild type, nearly one half the cases showed a marked regression of all the symptoms except the residual paralysis. Five per cent of the cases in New York and ten per cent of those in Massachusetts showed complete recovery. The mortality in this epidemic showed about the same percentage as the recoveries.—K.]

Treatment.—[Flexner and Lewis suggest the use of a nasal douche of one-per-cent peroxid of hydrogen in perhydrol as a preventive measure after exposure. Attempts at immunization, they think, should be directed to lessening the spread of the disease in the central nervous system, and offsetting the toxic action on the ganglion cells. The Massachusetts State Board of Health recommends that all cases should be quarantined, and that the urine, stools, and sputum should be disinfected.—K.] If we have an opportunity, even during the initial stage of the disease (when, of course, the diagnosis cannot usually be made with certainty), to attack the disease by our treatment, we may prescribe cold compresses or an ice bag to the head, and eventually, when there is high fever or great stupor, a tepid bath with cool affusions. We are but rarely led to try bloodletting by leeches behind the ears or on the temples, when there are signs of marked cerebral hyperæmia. Internally we usually prescribe a mild "intestinal derivative," such as 0.5 or 1 gr. (gm. 0.03 to 0.05) of powdered calomel every two or three hours, infusion of senna, etc.

After paralysis appears, we may expect the most success from electrical treatment, kept up consecutively for months, and, with interruptions, for years. We put a large, broad electrode on the vertebral column at the spot which corresponds to the place of the lesion in the spinal cord—on the cervical vertebræ in paralysis of the arm, and the lower dorsal in paralysis of the leg—while the other electrode serves for peripheral application to the paralyzed nerves and muscles. In this way we apply a moderately strong constant current, reversing it occasionally, for two or three minutes, partly stabile and partly labile, passing the cathode, or eventually the anode, slowly over the paralyzed muscles and nerves. We may also employ occasional interruptions and reversals of the current. Duchenne has also found persistent treatment by the faradic current of advantage. The sittings should take place three or four times a week, and later even oftener, if possible.

Besides electrical treatment, methodical gymnastic exercises of the muscles, that can still be moved to some degree actively, may also be of distinct advantage. [In many cases it is beneficial to have the gymnastic movements made in a warm bath, as the water buoys up the paralyzed limbs and renders movements easier.—K.] Regular and persistent massage of the muscles is also to be recommended in the later stages. In practice we cannot avoid prescribing certain embrocations, such as spirit of camphor, spirit of mustard, or spirit of formic acid. Passive motion is very important to guard against contractures, and to improve the already existing deformities.

The use of baths, of brine or ferruginous waters, is to be recommended, although, of course, they must not be overvalued. Brine baths and artificial carbonic-acid baths can be used at home. If circumstances permit sending the child to a bath during the summer months, we should chiefly consider the brine baths at Reichenhall, Kreuznach, Kösen, and Colberg; the acid salines at Rehme, Nauheim, and Soden; and eventually, with weak and anæmic children, the use of the iron baths at Pyrmont, Elster, or Schwalbach. Good results are sometimes obtained at the indifferent thermal baths at Teplitz, Wildbad, Ragatz, or Gastein. We also obtain good results, especially with older children, at the cold-water cures.

Very little is to be expected from the use of internal remedies. Iodid of potassium and strychnin are recommended, the latter in the form of subcutaneous injections, $\frac{1}{60}$ to $\frac{1}{20}$ gr. (gm. 0.001 to 0.003) daily.

When the acute process is wholly past and we can no longer count on any further regenerative process, the mechanical surgical treatment of the patient may often be of material service. In the first place the power of movement may be improved by orthopedic appliances, and troublesome contractures may also be relieved by tenotomy. The therapeutic attempts to join the tendons of muscles which can still contract to the tendons of the paralyzed muscles, in order to render it once more possible to obtain the effect of movement of these muscles (Nicolodoni, Vulpius, and others), are of especial interest. The symptomatic benefit thus obtained is very considerable. Details as to all the orthopedic and surgical methods of treatment are to be found in the special treatises. When we can no longer hope for any further improvement in the paralysis worth mentioning, the treatment is limited to improving the patient's general condition and strengthening him as much as possible by proper food and good air.

2. ACUTE POLIOMYELITIS OF ADULTS

(Acute Atrophic Spinal Paralysis of Adults)

Although it had been believed for a long time that the form of acute atrophic spinal paralysis, just described, occurred only in children, later observations by Moritz Meyer, Duchenne, Erb, F. Schultze, F. Müller, and others, have established the fact that precisely analogous cases of disease may also develop, although far less frequently, in adults, especially in young persons under thirty. There is no longer any doubt of this fact, especially if we consider an authentic anatomical lesion found by F. Schultze, and I have myself repeatedly observed undoubted cases of acute poliomyelitis in adults. [About five per cent of the Massachusetts cases in the recent epidemic were over twenty years of age. Ten cases occurred between the ages of thirty and seventy-two.—K.] We have, however, once before had to say that for a long time we have regarded the diagnosis of acute, and, as we shall soon see, of chronic poliomyelitis also, as too lightly made, and that certainly very many of the cases diagnosticated and published as poliomyelitis are to be classed as primary neuritis (*q. v.*)

The type of acute poliomyelitis of adults, so far as it has been established by definite observations made up to the present time, is not materially different from the type of the spinal paralysis of children.

We often cannot make out any ætiological conditions; sometimes exposure to cold, overexertion, etc., seem to favor the development of the disease. Especial importance must be attached to preceding infections of a mild type (sore throat, enteritis, etc.). The affected individuals are comparatively young. Cases are seen more frequently in men than in women.

The disease likewise begins with quite severe initial symptoms, fever, headache, somnolence, delirium, and vomiting, which may last from a few days to a week or two. The violent spontaneous pains in the extremities, which are frequently reported as a symptom of the disease, usually belong probably to those cases in which a primary neuritis, but not a poliomyelitis, is the anatomical lesion; but severe pain in the back [and limbs] may also occur in genuine poliomyelitis in the first acute stage of the disease. In some cases the initial general symptoms are insignificant. The disease begins at once with rapidly spreading paralysis. The paralysis develops with varying distribution, usually in single spurts, but always rather rapidly. The paralyzed muscles are perfectly flaccid, the cutaneous and tendon reflexes are wholly absent, and very soon a pronounced atrophy and reaction of degeneration appear, while the sensibility and the vesical and sexual functions remain normal.

The distribution of the paralysis can generally be studied much better in adults than in children. The extent of the paralysis may vary to a marked degree. It rarely affects all four extremities; generally it occurs in the form of paraplegia, or even of monoplegia. In the affected extremities we often find certain groups of muscles paralyzed, to which E. Remak first called attention. This is explained by the arrangement of the spinal motor centers. Thus, for example, it is worthy of note that, in paralysis of the crural region, the sartorius often remains entirely free; that in the leg the tibialis anticus, on the one hand, and the peronei and the extensor digitorum on the other, may be separately diseased; that in the forearm the supinator longus, supplied by the radial nerve, remains free, while all the other muscles on the extensor side of the forearm are paralyzed ("forearm type" of E. Remak); and that, on the other hand, the supinator may be paralyzed alone or together with the biceps, brachialis anticus, and deltoid ("upper-arm type" of E. Remak). By determining accurately what muscles are affected, we can determine the location of the disease in the cord according to the statements previously made in regard to the position of the spinal-motor centers. As has been previously stated, in addition to the extremities, we should never neglect to examine separately the muscles of the trunk.

In regard to *diagnosis,* especial attention must be paid to the distinction between poliomyelitis and neuritis. The greatest stress is to be laid on the initial pains and any other slight disturbances of sensibility, and the asymmetrical localization of the paralysis in poliomyelitis in distinction from the usual bilateral symmetrical localization of the polyneuritic paralysis. The fundamental difference in the two clinically closely related diseases has been previously emphasized. It seems to me, however, that the principal distinction is that in genuine circumscribed poliomyelitis there is an actual local focus of inflammation, but in polyneuritis there is a toxic (hematogenous) degeneration of the peripheral motor neurons. In the latter case, of course, the motor-ganglion cell itself may also be destroyed if the disease be severe, but this would always be a different process from the local poliomyelitic inflammation.

The *prognosis* is not wholly unfavorable, as complete recovery has been observed in isolated cases, although only after months. It is possible, however, that these were cases of polyneuritis. As a rule, in the poliomyelitis of adults, as in spinal paralysis of children, the same permanent paralyses, with atrophy and contractures, may be left after the subsidence of the acute inflammatory symptoms. If the muscles of respiration (intercostals, diaphragm) are affected, poliomyelitis is directly dangerous to life.

The *treatment* follows the same rules that we have mentioned in the spinal paralysis of children. The internal or subcutaneous use of ergotin may be added on the recommendation of some physicians.

3. SUBACUTE AND CHRONIC POLIOMYELITIS

(*Subacute and Chronic Atrophic Spinal Paralysis. Paralysie générale spinale antérieure subaigué* [Duchenne])

Our anatomical knowledge of the occurrence of a subacute and chronic poliomyelitis, in the sense of the authors who have described it, is still fairly imperfect. Confusions with multiple neuritis are also undoubtedly common here, and the diagnosis is not incontestable in all cases published under the name of "subacute poliomyelitis"; but there can be no doubt, from some recent and perfectly accurate observations (Oppenheim, Nonne, and others), that extensive subacute and chronic diseases may occur in the anterior horns of the cord and lead to the development of extensive paralysis. The ætiological and general pathological conception of these cases is, of course, still wholly unsettled. We are probably dealing with infectious toxic causes, which have a noxious action on the spinal motor ganglion cells.

In the cases classed under this heading a paralysis, first of both legs and somewhat later usually of both arms, develops in a comparatively short time —in the course of some weeks or months, at most. It usually has no special cause or any severe initial symptoms.

In some cases an accidental cause seems to have favored the outbreak of the disease. The onset of the disease has been observed repeatedly to follow an injury. The patient complains at first of weakness in the legs; he can no longer walk, and he is confined to the bed. A short time later the same disturbances appear in the arms, and lead to a more or less complete paralysis. More rarely the opposite condition obtains, that the arms are affected first and the legs later.

Many differences are observed with reference to the extent of the disease. Occasionally the paralysis is limited to the upper or lower extremities; it is even possible that only one limb is affected. There are many differences also in the rapidity with which the disease progresses (subacute and chronic cases). But the sensibility always remains wholly unaffected, except at most for slight paræsthesia, and there is also no pain, either spontaneous or occurring on pressure. Soon after the paralysis an equally extensive atrophy develops, and a distinct loss of electrical excitability, running parallel to it, which passes over into a partial or, in all severe cases, a complete reaction of degeneration.

I consider the fibrillary and fascicular contractions, which are almost invariably observed, as very characteristic and of great diagnostic value. In my opinion these contractions, which are occasionally very active, are the direct result of irritative conditions in the diseased spinal motor ganglion cells. Contractions of this sort do not occur in polyneuritis, and only rarely in acute poliomyelitis, since a rapid complete destruction of the ganglion cells occurs in this disease. The cutaneous and tendon reflexes are very much diminished and often entirely lost. The bladder and rectum, however, remain intact, and bedsores never develop if the patient has proper care. We sometimes notice a striking diminution of the sweat secretion. In rare cases the muscles of the neck, the lips, the tongue, and the pharynx are attacked by the disease.

After the paralysis has reached its greatest extent there is occasionally a pause. The condition remains stationary for months sometimes, and then a gradual improvement begins, which perhaps may go on to complete recovery, but generally the recovery remains incomplete; so that the patient has a more or less marked disturbance of function for life. The "middle form of chronic poliomyelitis," described by Erb, in which there is only a partial reaction of degeneration in the paralyzed muscles, almost always gives a good prognosis. The disease takes an unfavorable course if the respiration be impaired through involvement of the respiratory muscles, or, if the patient cannot swallow, through involvement of the muscles of deglutition, or if inhalation pneumonia set in. The autopsy, in cases of this class, shows extensive degenerative inflammatory changes in the anterior gray columns of the cord with a great destruction of ganglion cells, while generally only very slight changes (secondary degeneration) can be seen in the peripheral nerves.

As a consequence of what has been said, we shall be compelled to make a *diagnosis* of chronic poliomyelitis, when extensive paralysis comes on with subsequent atrophy and electrical reaction of degeneration and without any neuritic symptoms (pain!), and without disturbances of sensation or of the bladder. It is distinguished from "spinal muscular atrophy" proper by the fact that in this there is a slow atrophy, attacking fiber after fiber, so to speak, and that the atrophy and muscular weakness go absolutely parallel, while in chronic poliomyelitis the paralysis precedes the atrophy, and whole muscles or muscular regions are affected from the start.

The treatment is not absolutely hopeless, as is shown by the above description. But, at any rate, in all cases which show a continued progression of the paralysis, a very grave prognosis must be made. Treatment consists only in the employment of electricity, baths, careful massage, etc.

CHAPTER XI

THE SO-CALLED ACUTE ASCENDING SPINAL PARALYSIS

(Paralysis Ascendens Acuta. Landry's Paralysis)

In the year 1859 Landry described a disease under the name of "*paralysie ascendante aiguë*," which is chiefly characterized clinically by the fact that first the lower, and soon after the upper extremities, and finally a number of the muscular regions supplied by the medulla are attacked by a rapidly advancing paralysis, while the sensibility and the functions of the bladder and rectum remain normal. In many cases the disease terminates fatally. Examination of the nervous system has so far, however, shown no uniform anatomical lesion which is characteristic of all cases (*vide infra*). However, the clinical picture of Landry's paralysis is so striking that a comprehensive description of it seems demanded.

General Symptomatology.—Acute ascending paralysis attacks chiefly persons previously strong and healthy, in youth or middle life, somewhere between twenty and thirty-five years of age. Some cases also have been seen in children and older people. The disease seems to be more frequent in men than in women.

The affection almost always begins with certain prodromata. These consist of general malaise, moderate febrile symptoms, headache, loss of appetite, and quite frequently of dragging and tearing pains in the back and the extremities. After these symptoms have lasted some days, or more rarely some weeks, during which they are either comparatively slight or so severe that many patients are already confined to the bed, there usually comes on quite suddenly, or sometimes more gradually, a paresis, first of one leg, but very soon of both legs, which rapidly increases, and usually in a few days leads to an almost complete motor paraplegia.

The paralysis is flaccid in almost all cases. The legs may be moved passively without any muscular resistance, and the muscles show neither active nor reflex tension. Their electrical excitability remains perfectly normal in many cases, but there is sometimes a rapid loss of faradic muscular excitability. It is not yet proved whether complete reaction of degeneration occurs. The reflexes, both cutaneous and tendon reflexes, seem to be diminished or wholly lost in the majority of cases, but some exceptions to this rule have been known.

Sensibility is sometimes perfectly intact, but slight alterations do occur, and quite rarely there may be even marked anæsthesia. At times a noticeable delay of sensation is observed. We find no changes in the nerves of special sense. There is occasionally a slight œdema in the legs, which is perhaps to be regarded as a vasomotor disturbance. The marked sweating, from which many patients suffer, is also worthy of mention. The bladder and rectum in most cases are not at all affected, or they present merely slight and temporary disturbances.

A short time after the legs are attacked the arms also begin to be paretic. Marked motor weakness appears first in one, then in the other arm, and this may also increase to almost complete paralysis. The sensibility, the reflexes, and the electrical excitability show conditions like those in the lower extremities. The muscles of the trunk are also affected at the same time as, or still earlier than, the arms. The patient can no longer sit up in bed, turn on his side, or make similar movements. In some cases a paralysis of the muscles of the neck has also been observed.

The third and last stage of the disease is characterized by the appearance of respiratory disturbances and bulbar symptoms. Manifest signs of a beginning respiratory paralysis appear; the respiration is labored and difficult, the movements of the diaphragm are impaired, and the paroxysms of coughing grow weaker. Disturbances in swallowing, difficulty in articulation, and paresis of the soft palate and the lips may set in. In a few cases a facial paralysis and disturbances of the ocular muscles have been observed. The condition grows worse acutely, and, as we have said, in many cases death ensues.

Besides the symptoms thus far mentioned, referable to the nervous system, we find certain other symptoms in almost every case, which are less striking, but yet of greater significance in judging of the disease. The first of these is fever. The temperature is usually elevated from the beginning; it may temporarily show a considerable increase, up to 104° F. (40° C.), and later it varies between 100° and 102° F. (38° to 39° C.), but between times it may even sink to normal. Of the internal organs the spleen shows the most frequent changes. It is usually swollen moderately, but appreciably. There is also sometimes a slight albuminuria.

In the cases with a fatal termination the whole duration of the disease is sometimes only a few days, and, as a rule, a week or two, or rarely more. Fortunately, however, all cases do not terminate fatally. The disease may come to a standstill at any time, even if the most threatening symptoms be present. Then the paralysis shows no further advance, the disturbances present disappear, and recovery ensues after a course of several weeks. It is, of course, usually quite a long time before the patient again feels himself in possession of his full powers.

Pathological Anatomy and Pathogenesis.—If we consider the whole picture of acute ascending paralysis, the idea is necessarily forced upon us that we have to do here with an acute infection or autointoxication of the body, with a predominating localization in the motor nervous system. The beginning of the disease with general malaise corresponds perfectly to the prodromal stage of many other acute infectious diseases. The fever, the acute splenic tumor, and the occasional albuminuria also can scarcely be explained in any other way, according to our present views, except by the above hypothesis. There are, of course, no definite and positive data as yet in support of this hypothesis. Further investigations as to the anatomical changes in "acute ascending spinal paralysis" are still very desirable. In the earlier observations there is usually the statement of an "absolutely negative anatomical examination"; but these statements usually refer only to the spinal cord. The peripheral nerves were formerly not examined at all; and yet, as we have long proclaimed and as is now quite generally accepted, it is extremely probable from the clinical symptoms (pain, loss of electrical excitability, condition of the reflexes) that many cases of so-called Landry's paralysis are multiple neuritis (*q. v.*), and are nothing more than the most acute and most extensive form thereof. Some recent anatomical investigations have already confirmed this hypothesis.

It would, however, be incorrect to identify acute ascending paralysis completely with multiple neuritis, for in some observations (R. Schulz and F. Schultze, von den Velden) extensive acute myelitic changes have also been found in the motor portions of the spinal cord (lateral columns, anterior gray columns). The disease may then, perhaps, be interpreted as the most acute and extensive variety of acute poliomyelitis. At any rate, we must wait for many more observations and not decide prematurely upon the nosological position of the disease and a possible division into several varieties. [Recent investigations show that Landry's paralysis is an affection of the whole peripheral motor neuron, but that the ganglion cells in the anterior horns show, as a rule, greater changes than the peripheral nerve fibers.—K.]

Diagnosis and Prognosis.—In every paralysis of the lower extremities beginning acutely and accompanied by general symptoms and fever we must consider the possibility of an acute ascending paralysis, but only the further course of the disease can decide the question. Inasmuch as merely a well-characterized clinical group of symptoms is meant by the above term, the diagnosis is always easy to make, with attention to the peculiarities given above. It is more difficult, however, to decide accurately whether the case corresponds rather to the type of an acute multiple neuritis or to the type of an acute ascending spinal paralysis. We can judge as to this point only by careful attention to the single symptoms, especially the condition of the sensibility (pains, anæsthesia), of the reflexes, and of the electrical excitability.

The prognosis must at first be made with great reserve, and we must especially bear in mind the possibility of a rapidly fatal termination. If the first acute stage pass off fortunately and there be a decided cessation in the extension of the symptoms of paralysis, the prognosis is quite favorable, for we may then expect that the patient will be completely restored. [In many of the cases that do not terminate fatally there is a residual paralysis and atrophy similar to poliomyelitis.—K.]

Treatment.—We cannot be certain whether an energetic "derivative treatment" is of advantage in the beginning of the disease. Dry cups along the vertebral column are recommended, and even the use of the hot iron to the back. We would hardly advise the latter. It may be advisable, however, to prescribe inunctions with mercurial ointment, 30 to 45 gr. (gm. 2 to 3) a day, as in antisyphilitic treatment. At the onset of the disease careful sweating is also to be recommended. Of internal medicines, we should try at the beginning aspirin or salicylate of sodium in large doses (30 gr. [gm. 2] of salicylate of sodium three times a day), and also antipyrin. We may also try extract of ergot (ergotin), strychnin, and perhaps iodid of potassium. It also seems to be a good plan to begin galvanic treatment early, applying it to the spine and peripherally. If alarming attacks of respiratory insufficiency occur, electrical excitation of the phrenic nerve and of the respiratory muscles sometimes affords relief to the patient.

If the symptoms be arrested, electrical treatment and the use of baths may do most to hasten convalescence.

CHAPTER XII

NEW GROWTHS OF THE SPINAL CORD AND OF ITS MEMBRANES

Pathological Anatomy.—Tumors of the spinal cord are not particularly rare. The commonest primary new growth in the spinal cord itself is the glioma, which arises from the neuroglia, and forms a cellular and vascular tumor. This is situated most frequently in the cervical or upper dorsal cord; occasionally, also, in the lumbar enlargement of the cord. It often develops at the beginning in the posterior half of the cord, but later it may attain a considerable length and a transverse diameter of several centimeters, so that the spinal cord in the part affected may have a considerable tumor-like thickening, or it may even be changed throughout almost its whole length into a thick, shapeless mass. Inside the glioma we sometimes find secondary hemorrhages and (myxomatous) softenings. Central gliosis is distinct from real glioma. The former never leads to any special enlargement of the cord (tumor formation), but to a gliomatous degeneration, and especially to an abnormal cavity formation in the cord, particularly in a longitudinal direction. However, transitional forms between gliosis and real glioma probably occur. We will speak of central gliosis, this important and by no means very rare disease, more fully in a subsequent chapter.

Of other rare new growths in the spinal cord we may mention solitary tubercles, syphilomata, and sarcomata. Solitary tubercles develop occasionally in conjunction with a tubercular caries of the spine. They are generally located in the central gray matter of the posterior columns. Cysticerci have also been repeatedly observed in the spinal cord.

In the spinal meninges have been found sarcomata, endotheliomata, fibromata, lipomata, psammomata, myxomata, and syphilomata. The cases of extramedullary tumors observed by me were most frequently psammomata (generally fairly circumscribed growths, the size of an olive, which compressed the spinal cord from without), flat endotheliomata, arising from the inner surface of the dura mater, and sarcomata. Sarcoma of the dura has a great tendency to spread as a flat growth, so that it may finally surround a considerable portion of the cord with its mass of growth. Sarcoma is often multiple; thus it may coexist on the spinal dura mater and at the base of the brain, etc. As a rule, it does not invade the pia mater or the substance of the cord itself. There are also tumors arising from the pia, which up to the present time have been generally classed as sarcomata. According to more recent investigations, in a part of the cases, at least, we are dealing with extramedullary gliomata which originate from the outer gliomatous border of the spinal cord. Lipoma develops on the outer surface of the dura, starting from the fatty tissue between the vertebral canal and the dural sac. A carcinoma arising from the vertebræ may also reach the spinal meninges by direct invasion.

We know practically nothing of the ætiology of most new growths in the spinal cord. In all probability there is an abnormal congenital predisposition of the tissues, which furnishes the germ for the subsequent development of the new growth. It is worthy of note that the onset of the first symptoms is very often preceded by an injury (fall on the back, etc.), which thus seems apparently to give the first impetus to the formation of a tumor.

Symptoms and Course.—New growths in the spinal meninges are located principally at the level of the dorsal cord. The first symptoms of the disease are therefore radiating pains, either persistent and burning or paroxysmal and neuralgic, and paræsthesiæ in certain root territories. There may also be local pain, stiffness and pressure in the back, etc. There is rarely any characteristic tenderness over the spine. If the compression of the posterior roots increase, anæsthesia is produced, the distribution of which points to an involvement of definite root zones. The anterior roots are more rarely affected by the neoplasm than the posterior roots. In the former case motor symptoms, muscular rigidity, and circumscribed paralyses develop. In general, anæsthesia and paralysis arise as pure root symptoms only when the disease is extensive, since, as was mentioned before, the complete division of one sensory or motor root does not cause complete anæsthesia or paralysis in any region. Although this first "neuralgic stage" is especially characteristic of meningeal tumors, it may nevertheless be occasionally entirely or nearly absent. Cases of this kind have been especially described by F. Schultze. The more the tumor compresses the spinal cord itself in its growth, the more pronounced the spinal symptoms become. Generally, the patient first notices a slowly increasing paresis of one leg, which gradually extends to the other. With the usual location of the tumors in the cervical or thoracic segment of the cord, the disturbances of motion generally show all the characteristics of a spastic paresis (rigidity, increased tendon reflexes, etc.). Finally, an almost complete paraplegia may develop. Sooner or later disturbances of sensibility are added; generally, at first, of posterior column type. For evident reasons certain indications of a unilateral lesion not infrequently develop—namely, a preponderance of motor symptoms on one side, and of sensory disturbances on the other. Vesical disturbances also generally supervene; in short, there develops a complete picture of compression of the spinal cord, such as we have already described and which need not be again discussed in all its details.

Diagnosis.—The diagnosis of the extramedullary tumor depends, above all, on a close observation of the course of the disease, the slow onset of the affection with root symptoms, and the gradual development of slowly progressive compression symptoms. Since attention has been directed to the not infrequent occurrence of extramedullary neoplasms, the correct diagnosis has been made in numerous cases. Practically, this is of the greatest importance, since the prospect of removal of the tumor by operation, and therefore for an occasional complete cure of the patient, is by no means unfavorable. Horsley, in 1889, was the first to remove a tumor of the spinal cord, which was correctly diagnosed and localized by Gowers, and since then a fairly large number of cases have been operated upon with success, but, of course, there have also been many failures. The differential diagnosis between meningeal tumor and compression of the cord, due to vertebral caries without Pott's boss, is occasionally very difficult. We must then pay chief attention to the general symptoms of tuberculosis (heredity, habitus, fever, continued high frequency of the pulse, other tuberculous affections, tubercular reaction). If a meningeal sarcoma occurs as a metastasis or as one manifestation of multiple sarcomatosis, the discovery of a sarcoma in some other place (such as the skin) may put us on the right track in our diagnosis.

In glioma of the cord itself the initial symptoms of sensory irritation may be almost wholly absent, or at least they may be only slight. A complicated type of spinal disease gradually develops, in which all the spinal symptoms may occur in the most varied combinations. The disease from the beginning sometimes exhibits the ordinary type of a transverse spinal paralysis, which will show the well-known differences, according to the seat of the lesion (*vide* page 319). In other cases, however, certain peculiarities in the type lead us to suspect the possibility of a tumor. Among them especially is the early asymmetry of the symptoms on the two sides. Since a tumor may at first be confined to one half of the spinal cord (which scarcely ever happens in myelitis), the signs of a unilateral lesion of the spinal cord (*vide infra*, Chapter XVII) are often observed in tumors in a more or less pronounced fashion. Only later in the course of the disease does the clinical type of unilateral lesion gradually change to the ordinary transverse paralysis. A certain alternation in the symptoms, improvements, and new and quite sudden changes for the worse, are also noticed at times with tumors, a condition which is probably to be referred to changes in the fullness of the vessels, or to hemorrhages in the substance of the tumor. The manner in which the disease progresses is also to a certain degree characteristic. If we find on repeated examinations evident indications that the spinal affection is slowly extending upward, especially if the cutaneous anæsthesia is moving upward and the paralysis is invading higher muscular territories, this condition is decidedly in favor of the diagnosis of tumor, since it is far less frequently observed in other forms of disease. We may also consider in diagnosis the fact that glioma often develops in the cervical cord, or at least gradually invades the cervical cord; consequently, sensory disturbances and paralysis occur in the upper extremities in addition to the paraplegia of the legs. This is of rare occurrence in myelitis. In general we wish to emphasize again, that in slowly developing paraplegias only tumors of the spinal cord, gradual compression, and multiple sclerosis need to be considered. The differential diagnosis of these diseases (which certainly is often very difficult) principally concerns us. A careful Röntgen ray examination frequently aids materially in the discovery of a primary disease of the vertebræ. Neoplasms of the spinal meninges, as isolated observations have shown, can occasionally, perhaps, be correctly recognized by the demonstration of specific cellular elements in the spinal fluid obtained by lumbar puncture. We need not repeat here the details of a topical ("segmental") diagnosis; they follow the general rules which hold for all lesions of the spinal cord (page 285); but we must once again remind the reader that total anæsthesia of a given cutaneous territory means not only an involvement of the corresponding posterior root, as given in the diagram, but also an affection of the neighboring roots, especially the two roots above. After determining the upper boundary of the anæsthesia, we should therefore always suspect that the upper boundary of the lesion in the cord is one or two segments higher than would correspond to the diagram, because the individual cutaneous territories are always in relation with several spinal roots. The same rule also holds when the segmental diagnosis is based upon the motor symptoms. The disregard of this relation, which was first clearly established by Sherrington, is the reason why formerly the location was usually put too low, even when the tumor itself had been correctly diagnosticated. In determining the upper boundary of the tumor, furthermore, we should consider not only the anæsthesia, but also the symptoms of sensory irritation. These very often manifest themselves above the anæsthetic zone and then point with considerable certainty to an irritation of the corresponding posterior roots by the upward growth of the tumor. The accurate determination of the position of the different spinal segments and roots in relation to the bodies and spinous processes of the vertebræ is also of great practical importance. In regard to this, we will refer to Fig. 154, page 293.

A tumor of the spinal cord may last for many years. In new growths of the meninges the symptoms of sensory irritation alone may continue for several years before there are any well-developed transverse symptoms. Other cases, of course, run a much more rapid course. The *prognosis* of tumors of the cord is wholly unfavorable unless there is a possibility of operative treatment; but, as we have said above, this presents itself in but few cases. Otherwise the *treatment* is merely symptomatic. If there is a suspicion of previous syphilis we should necessarily try inunction and the exhibition of iodid of potassium. In sarcoma arsenic in large doses is perhaps of some service.

CHAPTER XIII

THE SYPHILITIC AFFECTIONS OF THE SPINAL CORD

Ætiology and Pathology.—The affections of the spinal cord provoked by syphilis must be divided into two principal groups. The first group is formed by diseases in which a slow degeneration of certain systems of neurons develops in a somewhat obscure fashion under the influence of previous syphilitic infection (probably from the action of some toxin). These diseases are called most properly "*metasyphilitic*" (so-called metasyphilis). Chief among them, as we have already seen, is tabes dorsalis (the metasyphilitic degeneration of the posterior root neurons), and also, probably, certain rare cases of degeneration of the lateral columns, and of combined systemic disease in the lateral and posterior columns (*PyS*, *KlS*, and the columns of Goll). The second group of syphilitic diseases is to be classed as genuine tertiary syphilis in distinction from metasyphilis. These affections are characterized by the development of a specific gummatous new growth in the cord, especially in the membranes (*meningitis gummosa*) and in the blood vessels (*endarteritis syphilitica*). This is usually quite extensive and not infrequently associated with corresponding syphilitic disease in the brain. Since metasyphilis of the cord has already been discussed in another part of this work, there remains to be considered here only the specific syphilitic (gummatous) affections of the cord.

Indications of syphilis of the cord may appear in six months to a year after the infection; but they develop more frequently in from two to five years after the infection, or even later. In only a few cases does a circumscribed gumma form, which then causes the symptoms of an intramedullary tumor. Diffuse gummatous meningitis is far more common; this almost always arises from the spinal pia mater, and only exceptionally from the dura mater (page 302). In such cases the spinal cord seems surrounded, in an irregular fashion, but to a considerable extent, by a newly formed, vascular granulation tissue several millimeters thick. The meningeal blood vessels are also almost always much diseased, their walls are thickened by endarteritic new formations, and their lumen obliterated or filled with thrombi. The posterior border of the cord is usually most affected. The disease usually has a considerable longitudinal extent. The region of the dorsal cord is often most affected, but in other cases it may be the cervical or lumbar cord, or sometimes the region of the conus medullaris and the cauda equina. The spinal roots especially are often surrounded by a thick sheath of newly formed gummatous tissue. Plugs of newly formed tissue may extend from the pia mater into the substance of the cord, or the syphilitic new growth may extend to the vessels of the cord itself; in such cases secondary tissue changes (destruction of nerve tissue, proliferation of neuroglia) develop as a result of the defective blood supply of the tissue. In many cases secondary sequelæ also develop in the tissues of the cord, similar to those we have become acquainted with in compression of the cord from tuberculous new formations on the dura mater (*vide* page 300). It is also worthy of mention that in a few cases genuine gummatous disease may apparently be associated with metasyphilitic degeneration.

Symptoms and Course.—It is evident that syphilis of the spinal cord cannot present any single definite type of disease on account of the multiple localization of the anatomical changes. We may say rather that syphilis of the spinal cord is characterized by the irregularity and the often unusual combination of its symptoms, and its changeable and varying course. From the different combinations of symptoms that occur we may best distinguish, relatively speaking, the following two forms of the affection:

1. Chronic Gummatous Spinal Meningitis and Meningomyelitis.—The disease at first shows definite root symptoms, especially symptoms of irritation of the posterior roots—paræsthesia, hyperæsthesia, shooting pains, neuralgia. Associated with these we sometimes find well-defined local pain in the back or in the sacral region, pain on pressure and on movement of the vertebral column. Motor symptoms appear somewhat later, as a rule, sometimes motor-root symptoms (atrophic paralyses in definite muscular territories, for example, in the muscles of the forearm and hand on one side or in the sacral distribution, etc.); or more frequently motor symptoms from implication of the cord itself, spastic paretic symptoms in one or both legs, sometimes in disease of the cervical cord, in both legs and one arm. Associated therewith are various, but usually slight, disturbances of sensibility, vesical disturbances, etc. If the region of the posterior columns and posterior roots be chiefly affected, affections resembling tabes may develop (*syphilitic pseudotabes* of Oppenheim). Not very rarely unilateral disease may also present the more or less clearly developed picture of Brown-Séquard's unilateral lesion. It is, of course, impossible to describe exhaustively all the combinations of symptoms that may be observed.

The whole course of the disease is usually chronic and, as has been said, very variable. In mild cases we may sometimes see almost complete recovery under appropriate treatment, but this is unfortunately a rare event, since the secondary tissue necroses, the results of compression, obliteration of vessels, etc., are incapable of complete restoration. We see improvement quite frequently, especially with timely treatment, which finally passes into a stationary condition. We must be prepared for relapses, fresh exacerbations, and the onset of new symptoms, which may also be cerebral. The disease may last for many—even twenty—years. The affection becomes directly fatal through the occurrence of dangerous complications (pyelocystitis) or severe attendant symptoms (apoplexy), etc.

2. SYPHILITIC MYELITIS AND SYPHILITIC SPASTIC SPINAL PARALYSIS.—In many cases syphilis of the spinal cord exhibits from the outset the symptoms of a myelitic affection. This form probably arises generally from the vessels of the cord. The syphilitic endarteritis of the smaller vessels leads to a rapid or slow necrosis of tissue and the symptoms of spinal disease which necessarily result therefrom. The disease often develops as rapidly as an acute myelitis. A more or less complete paraplegia comes on within a few days. Only on careful inquiry do we sometimes learn that it has been preceded for some time by mild precursory symptoms (weakness, paræsthesiæ, etc.). In other cases the paraplegia develops more slowly, but yet within a few weeks or months. Since the anatomical lesion is very often in the dorsal cord, the paraplegia far most frequently shows the signs of spastic paralysis. Naturally, however, many minor differences in the clinical picture occur. Frequently the disturbances predominate in one leg, or the symptoms correspond approximately to a unilateral lesion of the cord. In other cases we may observe the clinical picture of a spinal hemiplegia. All these symptoms show many variations in intensity during the course of the disease. Marked improvements alternate repeatedly with new exacerbations. The not infrequent combination with symptoms of cerebral syphilis should also be noted (basal symptoms, apoplectic seizures, immobility of the pupil, etc.).

Erb has described a special form of spinal syphilis under the name of *syphilitic spastic spinal paralysis*. The latter is found especially in young persons of the male sex. Weakness, stiffness, and often a lively tremor (ankle clonus!) occur first in one leg and soon after in the other. The gait constantly grows more laborious, slower, and decidedly spastic-paretic. The sensory disturbances are at first very slight, and there is no pain. Later, on careful examination, slight changes in sensibility are, of course, often to be found. Disturbances of the bladder are also rarely very prominent, but they are often plainly existent in a slight degree from the beginning. The condition may improve, especially with timely treatment, but, for reasons already mentioned, a morbid condition may often remain stationary for years, or new symptoms even may ensue. The anatomical basis of syphilitic spastic spinal paralysis may consist of a somewhat diffuse lesion, which, however, is localized principally in the lateral columns of the upper dorsal cord. But, in other cases, a combined disease of the pyramidal tracts, the lateral cerebellar (*KlS*) tracts, and the columns of Goll, has been found to be the anatomical cause of the spastic symptom-complex (Nonne and others). In these cases we appear to be dealing with a metasyphilitic degeneration (*vide supra*) of the tracts in question.

Diagnosis.—So far as any diagnosis of syphilis of the spinal cord is possible, it is evident from what has been said. The chief rule is to consider the possibility of syphilitic disease in all spinal affections that are atypical from the beginning. If a previous infection be certain, or if, at least, it be rendered probable by the special antecedents (abortions, etc.) or other coincident symptoms (scars, bony enlargements, aortic disease, etc.), the diagnosis is the more warranted. Of course a previous infection often enough cannot be established, even when there is certainly syphilis of the cord, and, on the other hand, other forms of spinal disease may, of course, occur in those who have been previously infected. The most characteristic features of syphilis of the cord have been mentioned above. Similar types of disease may be produced by multiple sclerosis, by sarcomatosis of the spinal meninges, by tuberculosis of the vertebræ, etc. We must therefore consider these affections chiefly in the differential diagnosis. Sometimes the success or failure of specific treatment may be of significance in diagnosis. [The Wassermann reaction is also of service in diagnosis. It is positive in the blood serum, but negative in the cerebro-spinal fluid.—K.]

Treatment.—In every case, even where there is merely a suspicion or a possibility of syphilitic disease of the spinal cord, we should institute at once a thorough antisyphilitic treatment. I think the best treatment is inunction with mercurial ointment (gr. xlv to lxxv [gm. 3 to 5] daily). Other physicians recommend especially subcutaneous injections of corrosive sublimate, salicylate of mercury, calomel, etc. Calomel injections, in particular, are said to produce remarkably good results occasionally, even after inunctions have failed. The mercurial treatment is almost always combined with the internal exhibition of iodin preparations, especially iodid of potassium (at first gr. xx to xxx [gm. 1.5 to 2], and later still larger doses daily, preferably in gelatin capsules), iodid of sodium, or one of the newer iodin preparations, of which iodalbacid, iodipin, and sajodin seem to us the best. [It is often necessary to give iodid of potassium in much larger doses. If the diagnosis of syphilis be probable, it is advisable to begin with doses of half a drachm (gm. 2) or more three times a day, which should be increased rapidly until the patient is taking 5 or 6 drachms daily (gm. 20 to 25), and sometimes, as in cerebral syphilis, it may be necessary to push the dose to 1 or 2 ounces a day (gm. 30 to 60).—K.] If circumstances permit, it is well to combine inunction with a course at some bath (sulphur baths in Aix, Neundorf, also Tölz, Hall in Austria, Wiesbaden, etc.). Every antisyphilitic treatment must be kept up a sufficient length of time and repeated after certain intervals, as is the general custom in the treatment of syphilis. Besides the specific treatment, the other therapeutic methods which are efficacious in chronic spinal affections also have their appropriate use (warm baths, mineral baths, galvanization). Individual symptoms (neuralgic pains, vesical troubles) require special attention.

The properly conducted treatment of spinal syphilis may give results which are sometimes excellent and often at least satisfactory. We can understand from what has been said why our hopes are naturally only too often disappointed.

CHAPTER XIV

SYRINGOMYELIA AND HYDROMYELUS

Pathological Anatomy and Pathogenesis.—The formation of abnormal cavities in the spinal cord is due either to a dilatation of the central canal (hydromyelus) or to a peculiar process, which is called central gliosis, with the secondary formation of cavities (syringomyelia proper). The cases of genuine hydromyelus are recognized by the fact that the cavity is found in the middle of the cord, corresponding to the position of the central canal, and that its walls are covered by cylindrical epithelium. Slight degrees of hydromyelus, in which the dilated central canal has a diameter of 1 mm. or 1.5 mm., are not infrequently found. The dilatation usually extends over only one segment of the cord. Greater degrees of hydromyelus, where the central canal is dilated to a diameter of 0.5 cm. or 1 cm. and the longitudinal extent is greater, are much rarer.

In regard to the origin of hydromyelus, we may assume, according to the teaching of Leyden, that the cause is quite generally some anomaly of development in the formation of the central canal. The hypothesis of Langhans that processes of stasis in the central canal, such as compression of the upper end by a tumor in the posterior cranial fossa, may lead to hydromyelus, is certainly not applicable to the great majority of cases.

The conditions are different in syringomyelia, which is of much greater practical importance. In this affection, according to many investigators (Westphal, Simon, F. Schultze, J. Hoffmann, and others), the whole process apparently begins with a gliomatous new formation or transformation of tissue in the central portions of the spinal cord. This peculiar process, which probably starts in the ependymal cells about the central canal, is called central gliosis. It consists of a replacement of nerve tissue by fibrous glia tissue. The central gliosis, which extends toward either the posterior or the anterior horns, has a decided tendency to break down and thus to form secondary cavities, which unite with the central canal or remain independent, and these cavities may extend longitudinally in the central portions of the cord with numerous ramifications and projections. To this secondary formation of cavities we give the name of syringomyelia (*vide* Fig. 192). In this condition the central canal itself hardly ever remains normal; it is usually dilated and shows many projections, reduplications, and the like, circumstances

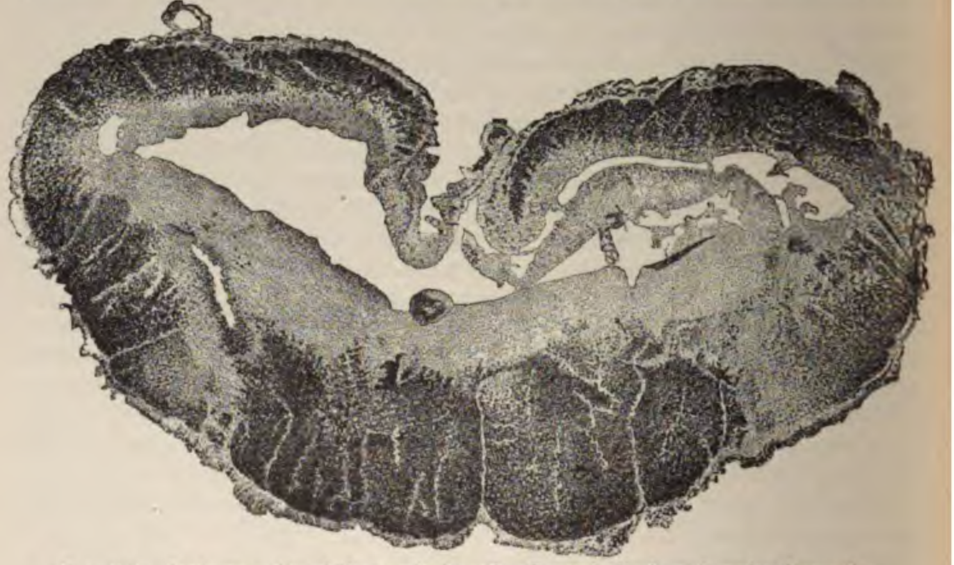

FIG. 192.—Syringomyelia of the cervical cord. Central gliosis and cavity formation. (From F. SCHULTZE.)

which clearly indicate that we must consider disturbances of development as factors in the origin of syringomyelia and central gliosis. We do not find any special enlargement of the cord in the nature of a tumor. Gliosis is thus distinct from true glioma formation (gliomatosis), but in some cases the distinction between gliosis and the formation of cellular glioma is ill-defined. In ordinary gliosis with syringomyelia, however, the cord appears normal externally, or, when the syringomyelia is extensive, it may appear flabbier, saccular, and flattened in the parts affected. Only on section do we recognize, even with the naked eye, the cavity formation in the center or to one side in the gray matter and extending into the horns of the cord. Under the microscope we find about the cavity the newly formed tissue, still proliferating peripherally and disintegrating toward the edge of the cavity. This tissue contains many newly formed ependymal cells in its central portion; toward the periphery the cells are fewer, and the tissue consists chiefly of a meshwork of fine glia fibers. We cannot say definitely to what circumstances we may refer the destruction of tissue and the cavity formation. Changes in the vessels, which are often found dilated, may perhaps play some part (hemorrhage, thrombosis, etc.). In most cases central gliosis and syringomyelia begin in the cervical cord and there attain their greatest extent, but they may extend downward in a lesser degree into the lumbar cord and upward into the medulla oblongata.

We must probably always look for the final cause of gliosis and syringomyelia in a defective congenital constitution of the spinal cord. This also explains the frequent combination of syringomyelia and hydromyelus, since both of these conditions are associated with abnormal embryonic development and are therefore coördinate with each other. Hydromyelus appears to me, however, to be usually a congenital stationary condition without marked clinical significance, whereas central gliosis with secondary syringomyelia is a progressive lesion, appearing first in later life, and producing a destruction of nerve tissue with consequent severe clinical symptoms of loss of function. Moreover, according to F. Schultze, injuries at birth in cases of difficult labor may perhaps also lead to hematomyelia and afford a basis for the later anomaly. Injury in later life is also frequently accused of producing the trouble; but we are probably always dealing in these cases with an abnormal congenital tendency, which develops only as a result of the trauma.

The disease is observed principally in early and middle life, being decidedly more frequent in men than in women. We have never seen it in children. It is very striking that syringomyelia, which is not a particularly rare disease among farm laborers, scarcely ever occurs among the well-to-do. Compared with the numerous hospital and dispensary cases, I can scarcely recall a single case in private practice! I have never observed the disease to be due to heredity, or to occur in several members of the same family.

Clinical Symptoms.—Slight degrees of hydromyelus or of syringomyelia, and even hydromyelus of fairly marked degree, may be met with at autopsies as accidental findings which have given rise to no clinical symptoms whatever. Other cases of extensive cavity formation give rise to complex forms of spinal disease, which usually escape correct interpretation during life. Thus we recall, for example, Späth-Schüppel's well-known case of general anæsthesia, a case under our own observation which presented the apparently pure type of spastic spinal paralysis, etc.

Opposed to such isolated cases, however, the great majority of cases of central gliosis and syringomyelia of the cervical cord present an unusually characteristic picture, so that we can often make a diagnosis with absolute certainty. Since the investigations of F. Schultze and Kahler we know that this type is by no means rare. It is therefore certain that it was often seen in the past, but it was usually falsely interpreted. The fact that the gliosis begins, as a rule, in the center of the cervical cord and that, as it progresses, it must always involve at first the gray matter of the posterior and anterior horns on one or both sides, is the reason why the group of symptoms always occurs in a way that is generally very characteristic, although it may show some variations. It is seen especially in men in youth and middle age (between twenty and forty).

The clinical symptoms of syringomyelia of the cervical cord develop very gradually, and almost always begin in the upper extremities—one arm usually being affected before the other. The symptoms observed in such cases may best be classed in three groups: 1. Motor weakness and muscular atrophy. These symptoms are dependent upon the involvement of the anterior horns in the process. The atrophy affects with especial preference the small muscles of the hand (interossei, thenar muscles), and also the muscles of the forearm, the deltoid, etc. Fibrillary twitchings and electrical reaction of degeneration are often found. The tendon reflexes are lost or else exaggerated, which may be easily explained from the special localization of the process. 2. Disturbances of sensibility are almost invariably associated with this muscular atrophy. The chief cause of these is always the involvement of the posterior horns in the process. The disturbances of sensibility almost always have this peculiarity, that the sensibility for pain and the temperature sense are at first always affected, while the sensibility for simple touch, muscular sense, and the pressure sense transmitted by the white posterior columns, may remain normal for a long time or throughout the disease. The fact that the patient is insensible to the influence of pain or heat is therefore of special significance, because it may frequently lead to wounds, burns, etc., on the hands and forearms, which the patient may often never notice, and which therefore may heal badly. The disturbances of the temperature sense may affect either the sensibility to cold or the sensibility to heat, or both at once. 3. The third group of symptoms is made up of peculiar trophic changes. The above-mentioned external injuries, caused by the analgesia, naturally play a large part in this connection (felons, bullæ, phlegmon, etc.);[1] but we must also assume, perhaps, special trophic disturbances to explain many processes, especially the peculiar thickening of the bones sometimes observed, the atrophy of the fingers, etc. In many patients there may thus develop a very peculiar and characteristic deformity of one or both hands, of which the accompanying cut (Fig. 193) gives a good idea. The hand and fingers are thickened, and some fingers are shortened by atrophy of the phalanges. The most striking abnormal vasomotor and secretory disturbances may also occur.

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[1] The so-called *Morvan's disease* is nothing but syringomyelia, in which these trophic and inflammatory changes are especially marked. Leprosy may produce very similar deformities of the hands. [The large number of cases coming under Morvan's observation in a small district in Brittany suggests, however, that Morvan's disease may be rather an infectious neuritis, possibly due to leprosy, and therefore distinct from a syringomyelia with marked trophic disturbances.—K.]

In many cases the symptoms remain confined almost entirely to the upper extremities. They progress so slowly that many patients are able to do a certain amount of work for a long time; but finally changes may also appear in the legs, either simple paresis and spastic conditions or symptoms similar to those in the arms already described. These arise only when the process involves the gray matter of the lumbar cord. On the other hand, the disease may extend upward from the cervical cord, and then very characteristic bulbar symptoms may ensue. The commonest bulbar disturbances are: partial loss of sensation in the region of the trigeminus, unilateral paralysis of the hypoglossus with unilateral atrophy of the tongue, paralysis of one vocal cord, unilateral paralysis of the accessory (weakness and atrophy of one trapezius and sterno-cleido-mastoid), paralysis of the soft palate, facial paralysis, abducens paralysis, etc. In many cases such bulbar symptoms are much more prominent than the spinal symptoms; in other cases they must first be looked for. If there be any suspicion of syringomyelia, we should never fail, above all, to make a laryngoscopic examination, because this very frequently establishes the diagnosis by demonstrating a unilateral paralysis of the vocal cords which could not be recognized in any other way. Furthermore, an accurate examination of the eyes is very important. Paralysis of the ocular muscles and nystagmus occasionally occur, without, however, having much significance. Disturbances of the sympathetic or ciliospinal center in the lower cervical and upper dorsal cord are much more frequent and therefore of diagnostic importance. The unilateral contraction of the pupil (myosis), associated with narrowing of the palpebral fissure and slight retraction of the eyeball on the same side, depends on the above cause. Dilatation of the pupil cannot be produced by the application of cocain. Coincident vasomotor disturbances of the corresponding half of the face also point with certainty to the sympathetic origin of the pupillary changes.

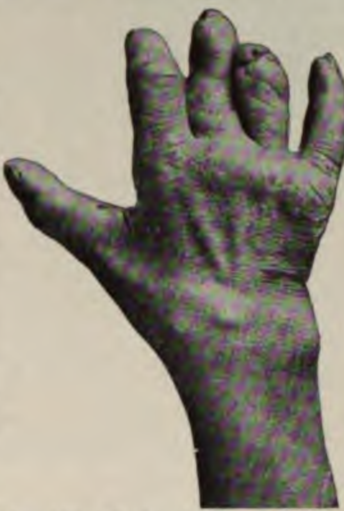

FIG. 193.—Syringomyelia of the cervical cord. Deformity of the hand, atrophy of the interossei and of the adductor pollicis. (Personal observation.)

It is a remarkable fact that syringomyelia is very often associated with a more or less pronounced kyphoscoliosis of the spine. In some cases the kyphoscoliosis depends undoubtedly on a unilateral paralysis of the muscles of the spine. In other cases, however, this explanation will not answer, and, in these cases, we believe that the scoliosis is an expression of the abnormal conditions of development and growth in the vicinity of the spinal cord.

Course.—The course of syringomyelia is very chronic, yet always unfavorable. Death finally ensues from some intercurrent disease, general weakness, pyelonephritis, or, as we have seen, from amyloid disease of the kidneys due to many suppurative processes. Sudden exacerbations of the symptoms may be produced by hemorrhages into the gliomatous tissue (occasionally due to trauma).

Diagnosis.—The diagnosis of typical cases of syringomyelia of the cervical cord is not difficult if we once know the type. A careful examination of the sensibility prevents us from mistaking it for spinal muscular atrophy. Any combination of muscular atrophy in the hands with analgesia and disturbances of the temperature sense must awaken the suspicion of syringomyelia, as must all unusual trophic disturbances occurring in the hands, frequent felons and phlegmons, etc. If the three groups of symptoms mentioned above coexist, we can be almost sure of our diagnosis. Only exceptionally can other processes in the cervical cord (such as compression following caries, as we have ourselves seen) produce a similar picture and give rise to confusion. The differential diagnosis between syringomyelia and leprosy may be more difficult, as the latter sometimes gives rise to a very similar type of disease; but in such cases the special endemic conditions, the peculiar changes in the skin, and finally the discovery of the specific bacillus, may enable us to make a correct decision.

Treatment.—There is no special treatment for syringomyelia. The treatment must be symptomatic. We should remember, when there is analgesia, to warn the patient of the possibility of injury.

CHAPTER XV

SPINA BIFIDA

(*Hydrorrhachis. Myelocele. Meningocele*)

We give the name of spina bifida to a congenital fissure-formation on the posterior side of the vertebral arches, due to anomalies of development, and associated with a hernia-like protrusion of the sac of the dura. The most frequent seat of the malformation is in the sacral and lumbar regions. Only rarely is the tumor so great as to hinder the birth of the child. Children afflicted with spina bifida are usually born normally, and not till afterwards do we find the tumor in the sacral region; its size may be from that of a small nut to that of the fist or larger. The skin above the tumor is sometimes entirely normal, but in other cases very tense and reddened. If we have an opportunity to examine the tumor carefully postmortem, we usually find beneath the skin the protruded sac of the dura, and beneath it the arachnoid. Only rarely is the dura also fissured, so that the sac is formed exclusively by the arachnoid. It is filled with a clear fluid which is precisely identical with the cerebro-spinal fluid. In rare cases there is also a dilatation of the central canal, hydromyelus; then the substance of the cord is atrophied to a greater or less extent, and the central canal communicates directly with the cavity of the spina bifida. In other cases the condition of the cord is normal; sometimes its lower end is adherent to one spot of the sac. We must refer to the text-books of pathological anatomy in regard to the many further details of the anatomy and the history of development.

In regard to the clinical symptoms of spina bifida, the condition of most children at first is perfectly normal, apart from the malformation. The tumor itself usually feels tense. If we exert pressure on it with the hand, we can often force part of its contents back into the vertebral canal. This causes an increase of the cerebral pressure, and we notice, besides the lessening of the spina bifida, a marked expansion of the fontanelles, and also the appearance of somnolence, twitching, and changes in the pulse and respiration, which demand a speedy interruption of this rather dangerous experiment. If such symptoms do not appear at all, we can conclude that the sac is completely constricted and closed.

Only rarely, however, does the child's condition remain normal. The tumor usually shows a slow growth, and the results of pressure on the spinal cord or on the cauda equina gradually appear. Paralysis, anæsthesia, vesical disturbances, bedsores, etc., develop, and these symptoms finally lead to death. Still more frequently the sac bursts, or its walls inflame, and this becomes fatal from the onset of a purulent meningitis.

The prognosis of most cases of spina bifida is accordingly to be regarded as unfavorable, unless we succeed in curing the disease by surgical treatment. Recovery has been brought about in many cases by methodical compression of the sac, and by puncture, with evacuation of the fluid and a subsequent injection of a solution of iodin to obtain an obliteration of the sac; but, on the other hand, the operative treatment of spina bifida is attended with many dangers, including liability to meningitis, so that we note frequent bad results as well as the favorable ones. We cannot here go into the details of the surgical methods for the cure of spina bifida; they can be found in full in the text-books of surgery.

CHAPTER XVI

SECONDARY DEGENERATIONS IN THE SPINAL CORD

ALTHOUGH the secondary degenerations in the spinal cord are chiefly interesting merely from an anatomical point of view, we must briefly describe them, because, in the first place, a clinical significance has been ascribed to them in certain quarters, and also because the study of secondary degeneration has been the starting point of all our present knowledge as to the systemic diseases of the spinal cord.

1. **Secondary Degeneration in the Spinal Cord After Cerebral Lesions.**—We already know (compare page 199) that every lesion of the great motor-ganglion cells in the anterior cornua of the spinal cord, and every permanent break in conduction in the motor nerves themselves is followed by a secondary degeneration of the peripheral portion of the motor fibers. We assume as the reason for this, as we have seen, a "trophic influence" of the said ganglion cells on the motor fibers arising from them, so that the latter degenerate when the conduction of that trophic influence is interrupted, or when the trophic-ganglion cells themselves are destroyed. Precisely analogous conditions exist for the first great portion of the motor-conducting tract, the lateral pyramidal tract, from the cortex cerebri to the anterior horns of the spinal cord (*vide*

Fig. 168, page 368). The great ganglion cells of the motor portion of the cortex cerebri also exert a trophic influence on the motor fibers arising from them, which extends to the end of these fibers—that is, up to, but exclusive of, the large motor-ganglion cells of the spinal cord. This atrophic influence may be understood if we regard the ganglion cell and its corresponding nerve fiber as forming one complete unit and properly only a single cell, a *neuron*, and that therefore the process of the cell body (the "nerve fiber") cannot possibly lead an independent existence when separated from the "ganglion cell"—that is, from the nucleus of the cell body. If, therefore, there be disease situated in the motor portion of the cortex cerebri itself, or in any part of the motor tract in the brain (the motor fibers of the corona radiata, the internal capsule, the crus, or the pons), by which disease the conduction is interrupted—if there be disease there, we repeat, a secondary descending degeneration of the motor fibers ensues in the whole portion below, down to, but exclusive of, the anterior horns of the gray matter. The secondary descending degeneration of the pyramidal tract is found correspondingly in the crus, in the pons, and in the pyramid of the same side as that in which the focus of disease in the brain is situated. From this point we can trace the main part of the degeneration below the decussation of the pyramids farther down the lateral column of the spinal cord on the opposite side (secondary degeneration of the lateral crossed pyramidal tract, see Fig. 194), while in many cases besides we find a slighter secondary degeneration in the anterior column of the spinal cord on the same side (secondary degeneration of the anterior uncrossed pyramidal tract). As we know from Flechsig's investigations, the relative amounts of the crossed lateral fibers, and the anterior fibers that remain uncrossed, vary in individual cases within certain limits. In the cases where no anterior pyramidal tract exists—that is, where all the motor fibers pass over to the lateral column of the opposite half of the spinal cord in the decussation of the pyramids of course a descending degeneration in the anterior column is wholly wanting. We must add, however, that in some cases a small number of fibers seem to proceed uncrossed in the lateral column, so that accordingly we may also have a slight secondary descending degeneration in the lateral pyramidal tract of the same (affected) side (Pitres).

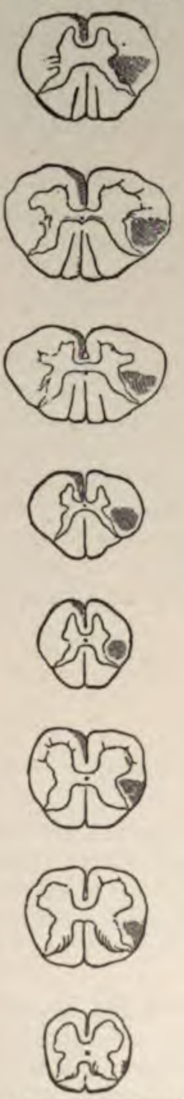

FIG. 194.—Secondary descending degeneration of the pyramidal tracts in a primary lesion of the left half of the cerebrum. The lateral pyramidal tract of the right half of the cord is degenerated down to the lowest part of the lumbar region (1 to 8); the anterior pyramidal tract of the left half of the cord is degenerated to the beginning of the lumbar enlargement (1 to 6).

2. **Secondary Degeneration in the Spinal Cord in Transverse Affections of the Spinal Cord Itself.**—If a lesion be situated in any part of the spinal cord, by which more or less of its transverse section is affected, the interruption of conduction in these fibers is also followed by the appearance of secondary degenerations which may be made out both in a descending and in an ascending direction (see Fig. 195). It is most frequently transverse myelitis, compression of the spinal cord, and tumors of the cord, which give rise to secondary degenerations. The latter, however, are, of course, never due to the kind of lesion, but only to its seat, and to the interruption of conduction caused by it.

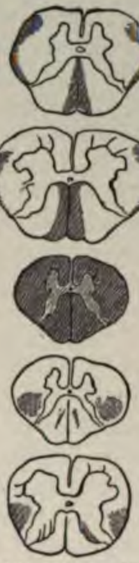
FIG. 195.—Secondary ascending and descending degeneration in a transverse affection of the upper dorsal region. The columns of Goll and the direct cerebellar tracts are degenerated upward. The lateral pyramidal tracts are degenerated downward.

The secondary descending degeneration affects the pyramidal tract in a fashion precisely analogous to that which we have also learned to recognize in secondary degenerations after cerebral lesions; but since the primary affection usually affects the pyramidal tract on both sides, the descending secondary degeneration, of course, develops in both lateral pyramidal tracts and also in the anterior pyramidal tracts, if they exist below the point of lesion. In general the area of the descending degeneration of the pyramidal tract is larger in transverse diseases of the spinal cord than it is in lesions of the brain. The principal reason for this is that, in spinal lesions, motor fibers arising from the lower centers (compare page 196), show descending degeneration. More accurate observations on this point are still to be desired. However, we have known for a long time (Lowenthal and others) that, in addition to the lateral pyramidal tracts, other fibers of the antero-lateral tracts are also affected. In the posterior columns, too, fibers with descending degeneration are found—in particular, a small comma-like bundle situated in the middle of each posterior column, and formed probably by descending collaterals of the posterior column fibers (so-called Schultze's comma tract; *vide* Fig. 195). This bundle, however, cannot generally be traced very far downward. The oval area in the lumbar and sacral cord which remains unaffected in tabes ("dorso-sacral longitudinal bundle") also shows descending secondary degeneration.

The secondary ascending degeneration, developing upward from the primary point of disease, affects several systems of fibers which conduct centripetally—the so-called columns of Goll (the internal portion of the posterior columns), and also two systems of fibers in the outer border of the lateral columns. The degeneration of the posterior columns is most extensive immediately above the point of interruption and gradually decreases upward (Fig. 195). We can readily understand this degeneration if we remember that the fibers of the posterior columns are the ascending fibers of the posterior roots, which have their origin in the cells of the spinal ganglia. Since the new fibers from the posterior roots which enter higher up are always external to the fibers coming from the lower roots, the degeneration must be limited more and more, as it ascends, to the region of the columns of Goll. A secondary ascending degeneration in the posterior columns also follows division of the posterior roots, and also, of course, injuries of the cauda equina. The secondary ascending degeneration in the lateral columns is made up of a smaller posterior and a broader anterior portion. The former is the lateral cerebellar tract discovered by Flechsig; its fibers arise from the cells of Clarke's columns in the lower dorsal cord, and therefore they must degenerate when separated from these cells. The fibers may be traced upward into the corpus restiforme, and then to the cerebellum. The bundle of fibers anterior to the lateral cerebellar tract, which also degenerate upward, is distinct from the lateral cerebellar tract in its development and its origin. It probably arises from cells of the posterior horns, and is called Gowers's tract or the anterolateral ascending tract. Its termination and significance are still unknown. We have observed an ascending degeneration of sensory fibers in the lateral columns after destruction of the posterior horns.

Although no clinical significance at all can be attributed to secondary ascending degeneration, the theory first advanced by the French observers (Charcot and others) still prevails to a great extent, that secondary descending degeneration causes definite clinical symptoms. The secondary contractures and the increase of the tendon reflexes in the paralyzed limbs, occurring in hemiplegia, are especially referred to the condition of irritability which the degenerating fibers have assumed. We shall see in a later section that this theory is by no means proved, and is even improbable, so that, in our opinion, secondary descending degeneration also has no material clinical significance.

3. **Secondary Degeneration in the Spinal Cord After Amputations.**—If we examine the spinal cord of persons who have had an arm or a leg amputated some time before their death, we sometimes find that the entire corresponding half of the cord is smaller; but the diminution of the posterior column on that side is the most frequent. On microscopic examination we can detect a slight but evident loss of fibers in the region of the column of Goll—that is, in the median portion of the posterior columns. We can also discover a slighter loss of fibers in the column of Goll on the other side (opposite to the amputation). We cannot enter into a more detailed description here of this condition, which is of interest theoretically, but which need not be considered practically and which, furthermore, is in many respects not fully explained.

CHAPTER XVII

UNILATERAL LESION OF THE SPINAL CORD

(Brown-Séquard's Spinal Paralysis)

UNILATERAL lesion is not a definite disease of the spinal cord, but a peculiar group of symptoms which occurs whenever an interruption of conduction is produced by any affection in one lateral half of the spinal cord. Since the symptoms in these cases were first carefully studied clinically and experimentally by Brown-Séquard, we often call the type of disease in question "Brown-Séquard's paralysis." We see this paralysis in its purest form in injuries of the spinal cord. Almost perfectly exact sections of one lateral half of the spinal cord have been produced by stabs from a knife, a sword, etc. Tumors and syphilitic affections especially, and more rarely central gliosis, multiple sclerosis, hematomyelia, compression of the cord in caries or cancer of the vertebræ, etc., may also, during a certain period of their course, cause the symptoms of a more or less sharply defined unilateral lesion.

The peculiar condition of the symptoms in unilateral lesion is easily explained by a consideration of the course of the fibers in the spinal cord. In the accompanying diagram (see Fig. 196) the motor fibers from the anterior roots are marked *v*, the sensory fibers from the posterior roots, *h*. As is well known, the sensory fibers, *h*, pass in part (*sk*) at once into the opposite half of the spinal cord, and accordingly decussate with the corresponding sensory fibers of the other side, while another part (*su*) ascend in the posterior columns without decussating. All the motor fibers, *v*, however, pass upward uncrossed on the side on which they enter the spinal cord, and mainly in the lateral column. If now, for example, there be situated on the right half of the spinal cord at *a* a lesion—e. g., a section of one half the cord—the conduction of those motor fibers which come from the right side is interrupted, as well as the conduction of those sensory fibers which come from the left side. From this it follows that there must be motor paralysis and anæsthesia in the area of innervation of the uncrossed sensory fibers on the same side of the body as the lesion in the spinal cord, and, on the other hand, anæsthesia of the area of innervation of the crossed sensory fibers, but no motor paralysis. If the affection be situated in the dorsal or lumbar cord, the leg on the corresponding side is paralyzed, and the leg on the other side is partly anæsthetic. If the lesion be situated in the cervical cord, above the entrance of the nerves for the upper extremities, the arm and the leg on the side of the lesion are both paralyzed (spinal hemiplegia), while the arm and the leg on the other side show certain sensory disturbances, but possess normal motility; but unilateral disease of the lowest end of the cord (the sacral cord and conus terminalis) may not give rise to the picture of a Brown-Séquard's unilateral lesion, because at this level no full decussation of sensory fibers has taken place. At most we find a flaccid atrophic paralysis in one leg with a disturbance of sensation on the same side, and an anæsthesia of the scrotum and penis on the other side, because the sensory fibers for this region enter the lowest part of the cord, and therefore have their decussation the lowest.

The motor disturbances in unilateral cord lesions correspond to the extent of the lesion in the affected half of the spinal cord. If all the motor fibers are interrupted, a complete paralysis in the area of distribution of the severed motor nerve tracts must naturally follow. Very frequently, however, the disease does not lead to a complete destruction of the affected half of the spinal cord, but corresponds more to the type of so-called hemiplegic paralysis (*vide infra*). In the leg the flexors are more affected, as a rule, than the extensors. On drawing the leg up, a coincident contraction of the tibialis anticus muscle (tibialis phenomenon, *vide supra*, page 393) then ensues. In addition, there are generally marked spastic symptoms in the affected leg (hypertonia of the muscles, increased tendon reflexes, etc.). If the anatomical lesion be located in the cervical segment of the cord, there is motor disturbance of the arm and leg of the corresponding side, and then we have, as already mentioned, a pronounced clinical picture of "spinal hemiplegia."

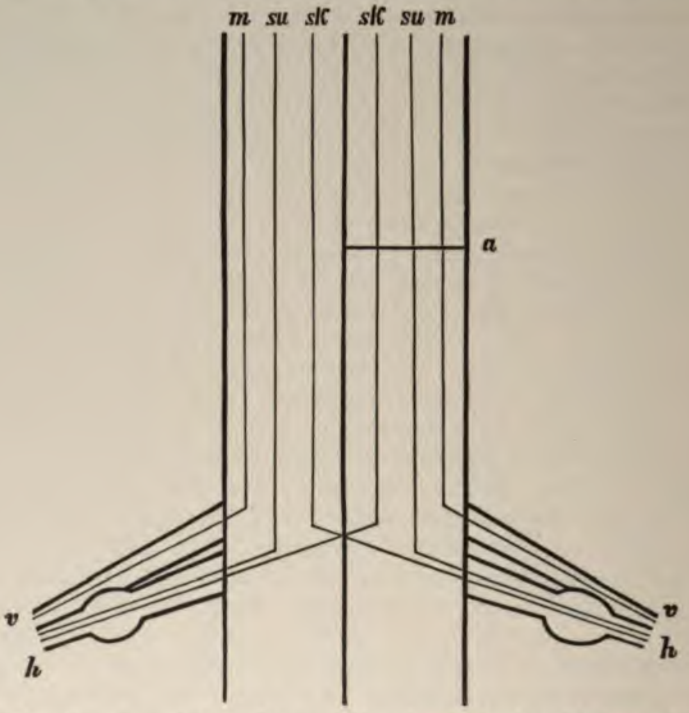

FIG. 196.—Schematic representation of the conditions in unilateral cord lesions. At *a* division of one half of the spinal cord; *v* = anterior root; *m* = motor tract; *h* = posterior root with spinal ganglion; *su* = uncrossed sensory tract (muscular sense, etc.); *sk* = crossed sensory tract (pain and temperature sense.)

The exact study of the sensory disturbances in unilateral cord lesions is very interesting. Corresponding to the preceding statements concerning the varying courses of the different sensory tracts, in the first place, we generally also find marked sensory disturbances on the paralyzed side, but mainly only in respect to the muscular sense (sensation for passive motions) and the deep pressure sense. Moreover, we consider it likely that in complete unilateral cord lesions the simple sensations of touch are also diminished on the affected side, or even entirely absent. However, further investigations must be made on this point. On the other (unparalyzed) side marked disturbances of the pain and temperature sense are found regularly, because the tracts for these forms of sensation decussate in the spinal cord. At a level corresponding to the lesion in the spinal cord itself a certain number of sensory fibers must naturally be interrupted before their decussation at their point of entrance into the spinal cord. For this reason, at the upper level of the sensory disturbance we also frequently find on the paralyzed side a small anæsthetic zone of skin which, at the same time, is also analgesic and thermo-anæsthetic. We occasionally observe a cutaneous hyperæsthesia to painful sensations (needle pricks) and occasionally also to thermal stimuli. This is hard to explain and is less constant than the sensory disturbances described above. This hyperæsthesia is sometimes found over the entire paralyzed leg, more frequently in a band-like zone at the upper level of the sensory disturbance. This symptom also requires further careful study.

It is difficult to make any general statements concerning the condition of the cutaneous reflexes. Frequently we find an absence of the abdominal and cremaster reflexes on the paralyzed side. Where spastic symptoms exist the Babinski [and Oppenheim] reflexes are generally present (reflex dorsal extension of the big toe). Finally, it must be remarked that in a unilateral lesion of the posterior columns, without material involvement of the anterior portion of the spinal cord, an ataxia of the leg and arm of the same side can be demonstrated. Vasomotor disturbances (increase of the temperature of the skin, etc.) have also been observed on the paralyzed side.

If other spinal symptoms (disturbances of micturition and defecation, neuralgic pains, muscular atrophy, changes in the electrical excitability, etc.) are associated with the symptom-complex just described, these symptoms are dependent upon the special localization of the disease; for we must take special note that the clinical picture of a unilateral lesion may not be perfectly distinct, but we can often recognize only a few prominent features. However, with the aid of our present knowledge concerning the course and function of the different spinal tracts, it is generally possible, by carefully utilizing all the symptoms found, to make at least an approximately correct focal diagnosis of the location and extent of the existing disease.

We need add nothing as to the prognosis and treatment of unilateral lesion, because, of course, they are governed entirely by the form of the primary disease.

III. THE DISEASES OF THE MEDULLA OBLONGATA

CHAPTER I

PROGRESSIVE BULBAR PARALYSIS

(Glosso-labio-laryngeal Paralysis)

Duchenne, in 1860, described for the first time in complete detail the symptoms of a disease to which Wachsmuth subsequently gave the name of progressive bulbar paralysis. Duchenne did not, however, recognize the true seat of the disease, and it was not until 1870 that Charcot in France, and E. Leyden in Germany, were enabled to confirm the suggestion of Wachsmuth that the lesion is a progressive degeneration and atrophy of the nuclei in the medulla oblongata. Since then our knowledge of the disease has rapidly increased, both from the clinical and the anatomical standpoints, and Kussmaul and others have thoroughly investigated its relations to two other closely allied forms of disease—amyotrophic lateral sclerosis and progressive muscular atrophy.

Ætiology.—We have scarcely any positive information about the cause of the disease. Heredity seems in general of slight importance; but some observations indicate that the disease may also occur in families, so that we must probably assume in the last instance that the special cause of the affection is usually a congenital abnormality of the system of neurons which later become diseased. In some cases its origin is ascribed to catching cold, emotional excitement, traumatic influences, or excessive bodily exertion. Perhaps it is sometimes occasioned by excessive use of the muscles to which the disease is chiefly confined, as in playing on wind instruments; but in many instances no possible external cause can be found. Men seem somewhat more liable to be attacked than women. The disease hardly ever appears until middle or old age—that is, after thirty-five. Only a few cases have been observed in childhood.

Clinical History.—The symptoms are almost always very slow in their development. There may be mild premonitory symptoms—such as painful sensations in the back of the neck. Then there is a very gradual appearance of difficulty in articulation. Many words are pronounced indistinctly. The first trouble is noticed especially with letters in the utterance of which the tongue plays an essential part: E, R, L, S, G (hard), K, D, T, and N.[1] It is easily seen that the derangement is not aphasic. There is no forgetting or confounding of the words or letters, but the innervation of the tongue has become impaired. Long before the ordinary movements of this member are visibly embarrassed, the patient has lost the ability to make those more delicate manipulations of it which are essential to normal speech. This disturbance of speech is termed articulatory disturbance of speech (alalia or anarthria).

By the time this has become somewhat marked, it is usually possible to detect, on close examination, that the tongue is beginning to atrophy. It seems flabby, thin, and less rounded. Here and there its surface presents furrows and depressions, and often the individual fasciculi exhibit active fibrillary contractions. Just as in progressive muscular atrophy, the impairment of motion usually keeps equal pace[2] with the atrophy. The greater the atrophy, the less is the mobility. Finally, it becomes quite impossible to project the tongue from the mouth or move it from side to side. The tongue lies flat and limp on the floor of the mouth. Its surface is often diversified with furrows and depressions, containing much desquamated epithelium or the like. Evidently any great impairment of motility in the tongue hinders not only speaking, but also chewing and swallowing. The organ can no longer bring out such portions of the food as get between the cheeks and the teeth, nor can it push the bolus backward within the grasp of the pharyngeal constrictors.

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[1] [Except as otherwise specified, the letters and words used as examples here and later on are to be given the ordinary English pronunciation.—K.]

[2] At the commencement of the disease the paralysis may possibly seem greater than the atrophy, so far as the latter can be detected. Nor would it be impossible for a primary lesion of the nuclei of nerves to result in a paralysis before the secondary atrophy of the muscular fibers had become completely developed, or for the degeneration to begin in the corresponding central neurons. On the other hand, it must be borne in mind that numerous individual muscular fibers in the lip or tongue may be already atrophied before the eye or the touch can appreciate any change in bulk.

Even before the atrophy of the tongue becomes extreme, analogous disturbances usually appear in neighboring groups of muscles. As a rule, the muscles of the lips are affected next after the tongue. The first thing the patient notices is a peculiar feeling of stiffness or tension in the lips. Movement becomes gradually more and more difficult, and the patient becomes unable to pucker his lips so as to whistle. Speech is also noticeably interfered with, for now all those letters the pronunciation of which demands labial movements are very imperfectly articulated, and at last they cannot be uttered at all. These are O, A (long), P, F, B, M, and V; and also the sound of double O, as in tool. It also becomes gradually evident that the lips atrophy. They grow thin, with sharp edges and wrinkled skin. Fibrillary contractions are not infrequently visible.

This atrophy of the lips (orbicularis oris) is occasionally followed by atrophy and paresis of some of the other muscles of expression supplied by the lower division of the facial nerve. The general facial expression of a patient with bulbar paralysis thus comes to bear a very characteristic stamp—the mouth remains half open, and seems to be broadened, the corners of the mouth are drawn down, and the lower lip hangs down, so that the whole aspect is persistently lachrymose. Even in laughing, the lower half of the face relaxes comparatively little; while the region supplied by the upper division of the facial nerve, and the movements of the eyeball, remain, as a rule, perfectly normal.

The third group of muscles affected are those of the pharynx and larynx. The soft palate becomes paretic, and produces further trouble in swallowing. Quite often the liquid ingesta are regurgitated through the nose. The voice becomes nasal. The production of many sounds, and in particular of B and P, is now impossible, since, in addition to the labial paresis, a portion of the essential current of air escapes through the nostrils. This explains why the letters mentioned can sometimes be pronounced better if the nose be compressed. The paralysis of the constrictors of the pharynx impedes deglutition more and more, till the impairment of nutrition becomes extreme.

The enfeebled action of the laryngeal muscles is betrayed, in the earlier stages of the disease, by a certain weakness and monotony in speaking. Modulations of the voice, and the production of the higher notes, as in singing, are no longer possible. If the innervation of the larynx becomes still more impaired, it becomes a very serious matter. If the arytænoid cartilages do not press together firmly on swallowing, the entrance to the larynx is inadequately closed, and food is often swallowed the wrong way. Liquid and even solid ingesta get into the larynx, and excite a violent cough; or, being inhaled into the air-passages, they cause bronchitis or lobular pneumonia. The paralysis may reach such a degree that the voice is at best a hoarse whisper. With the laryngoscope we can see that the vocal cords are paralyzed. The inability to close the glottis tightly is extremely unfavorable, for it renders the patient unable to cough vigorously. Mucous accumulations may therefore come to be the source of extreme dyspnœa.

The catalogue of symptoms is not yet ended. As we have seen, the muscular atrophy of the tongue and lips can invariably be detected. That of the pharyngeal and laryngeal muscles cannot be demonstrated during life, although it is to be found postmortem. Inasmuch as the process is one of neural atrophy, the affected fibers ought to give the reaction of degeneration to electricity; but this is difficult of actual proof, just as it is in progressive muscular atrophy, because numerous healthy fibers lie side by side with the degenerated ones. Still, in an advanced case, careful examination will usually bring out an evident degenerative reaction here and there in the tongue and lips.

The disturbance of reflex action is often striking. Usually the reflexes are greatly diminished or even absent, so that one can tickle the root of the tongue and the epiglottis without causing the patient to gag. In a few instances the facial muscles exhibit an increase of tendon reflex, as can be shown by tapping upon the tendons, the periosteum of the jaws, or the bridge of the nose. This behavior reminds one of the condition of the muscles in amyotrophic lateral sclerosis (*q. v.*).

Exceptionally, still other muscular groups are involved. Of such disturbances, the most frequent is in the region supplied by the motor branch of the trigeminus, impairing mastication. The impairment of these muscles now combines with the labial and lingual atrophy to render chewing almost impossible. In very rare cases the ocular muscles are also involved, with resulting ptosis and strabismus.

All the symptoms thus far enumerated are exclusively motor. Sensation is perfect to the end. The sensibility of the skin of the face and of the mucous membrane of the tongue and mouth, as well as the sense of taste, are unimpaired. Disturbances of sensation in the distribution of the trigeminus, and more or less deafness, have been reported in one or two cases, but there is some doubt about the observations. It does, however, seem certain that secretory and vasomotor derangements are frequent. Salivation deserves especial mention. In many cases of bulbar paralysis it is a constant symptom, so that the patient is obliged to keep a pocket handkerchief to his mouth to catch the fluid as it dribbles away. This is due, to a certain extent, to the fact that the secreted saliva cannot be swallowed, and, as the lips do not shut tightly, it naturally escapes from the mouth; but volumetric examinations have rendered it pretty certain that the amount of saliva is abnormally large. The explanation of this has not been determined. Nor as yet do we know much about the vasomotor disturbances. Many patients complain of a feeling of heat and "boiling" in the head. We may also mention in this connection that occasionally, toward the close of the disease, the pulse becomes very rapid (140 to 160). This is probably due to paralysis of the vagus.

The course of the disease is invariably chronic. The order in which the symptoms appear is, as a rule, that in which they have just been described. The atrophy and paresis appear first in the tongue, then in the lips and the neighboring muscles of the face, and lastly in the muscles of the soft palate, pharynx, and larynx. Still, there may be some deviation from this. Usually the progress of the disease is very gradual. There may be an apparent arrest of the trouble, or less often there are quite sudden exacerbations. When all the different symptoms are well developed, the clinical picture is unusually characteristic. The peculiar immobility of expression; the broad, slightly gaping mouth, with the atrophied lips; the almost unintelligible speech, low, monotonous and labored; and the inability to swallow—these often betray the disease at once. The last stage of the illness is the more distressing in that the intelligence remains to the end entirely unclouded.

The entire duration of the disease is usually several years—perhaps two to five. If death is not caused by some intercurrent trouble, it is brought about in one of three ways: either through inanition, due to the increasing difficulty of deglutition; through pulmonary complications—namely, bronchitis, lobular pneumonia, or gangrene, as a result of food passing down the trachea; or through sudden asphyxia or cardiac failure.

Pathology. Nature of the Disease, and its Appearance as a Symptom of Progressive Muscular Atrophy or of Amyotrophic Lateral Sclerosis.—If we seek the anatomical lesion corresponding to the group of symptoms above depicted, we shall find, on microscopic examination of the nervous system, in all cases of this description, a typical disease of the medulla oblongata. The ganglionic nuclei and the nerves (compare the accompanying anatomical diagram, Fig. 197), corresponding to those muscles which we have found to undergo atrophy in bulbar paralysis, present distinct evidences of degeneration. This is most readily demonstrated in the nucleus of the hypoglossus. The ganglionic cells have some of them entirely disappeared, while others are greatly atrophied. The connective tissue is increased in amount, and the walls of the blood vessels traversing the nucleus are thickened. In the earlier stages there are often many cells which contain granules of fat. The same changes, though perhaps less pronounced, are exhibited by the common nucleus of the vagus and accessorius, that of the facial, and sometimes also that of the glossopharyngeal nerve. The other nuclei are perfectly normal. We never find a diffuse "inflammation," but in every case a primary degeneration of the nuclei, which spreads no farther.

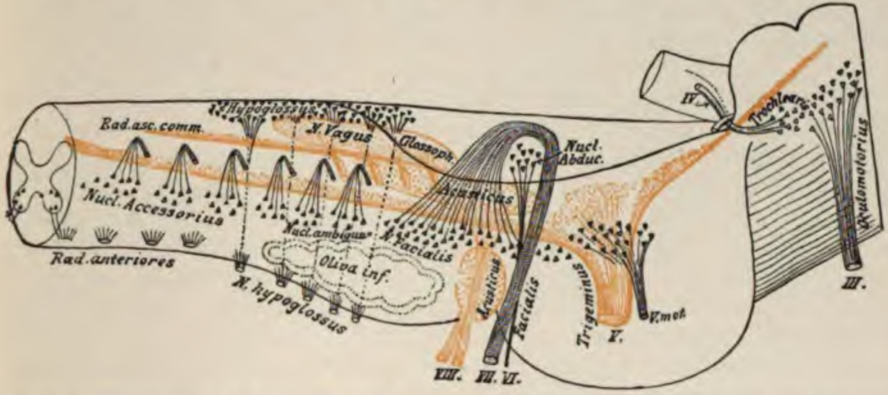

FIG. 197.—The position of the nuclei of the cranial nerves. The medulla oblongata and the pons are supposed to be transparent. The nuclei of origin (motor) are black, the terminal nuclei (sensory) are red. (From EDINGER.)

Starting from these nuclei, the degeneration and atrophy may be seen to extend into the nerve fibers which issue from them. The roots of the hypoglossus, vagus, accessory, and facial nerves can often be seen by the naked eye to be diminished in size and of a gray color. The microscope always shows a partial atrophy of their fibers. Finally, there is a corresponding atrophy of the muscles of the tongue, lips, and other parts. We need not enter into detail, for the histological conditions are precisely those seen in the muscles of the trunk and extremities in the spinal form of progressive muscular atrophy.

We find, therefore, that progressive bulbar paralysis is perfectly analogous with progressive muscular atrophy. The motor nuclei in the medulla oblongata are the motor and trophic centers of the bulbar nerves and of the muscles which these nerves supply. The relation is precisely the same as exists between the anterior cornua of the spinal cord on the one hand, and the spinal nerves and the muscles which they innervate on the other. In both diseases there is a degenerative atrophy of the individual neuro-muscular elements, the so-called neurons (consisting of nerve cells and peripheral nerve fibers), and their corresponding muscular fibers. In both diseases the atrophy and the functional disability of the muscles keep pace with each other, and in both the affection is limited strictly to the motor tract, sensibility suffering no impairment whatever. Certain questions about bulbar paralysis are as unsettled as similar ones about progressive muscular atrophy. It is uncertain whether the primary degenerative process is limited to the bulbar nuclei, and the degeneration of the nerves and muscles is to be regarded as secondary, or whether the entire motor apparatus, from the ganglion cell to the muscular fiber, is simultaneously attacked; or, finally, whether the atrophy begins possibly in the muscle and involves the corresponding nerve fiber and nerve cell only in its further progress. We think it improbable that these points will be cleared up very speedily. Their solution would seem to be only of theoretical interest.

We certainly must recognize, however, the essential identity of progressive bulbar paralysis and progressive muscular atrophy. The resemblance becomes even more striking if we consider that very frequently both diseases are present simultaneously. Often, after a case of progressive muscular atrophy has lasted for some time, the symptoms of bulbar paralysis also appear. On the other hand, an illness may begin with bulbar symptoms, and later on be complicated by atrophy of the muscles of the extremities—almost always seen first in the arms. If cases of this sort come to autopsy, we find a combination of the anatomical lesions of both diseases; in addition to the degeneration of the nuclei in the medulla oblongata, there is marked atrophy of the ganglionic cells in corresponding places in the anterior gray cornua of the spinal cord.

Progressive bulbar paralysis has as close a relation to amyotrophic lateral sclerosis (*vide* page 368) as to progressive spinal muscular atrophy. Amyotrophic lateral sclerosis is at first situated, as a rule, only in the portions of the motor-conducting system, including the pyramidal tracts, which supply the extremities; but, finally, there is very often an involvement of the motor bulbar nuclei—that is, bulbar paralysis sets in. It seems justifiable, therefore, to say that these three diseases—progressive bulbar paralysis, progressive spinal muscular atrophy, and amyotrophic lateral sclerosis—differing as they do in the localization of their lesions, are yet closely allied. They are to be regarded as manifestations of one morbid process which is probably essentially the same in its pathology and ætiology. We do not know the special cause of this process of degeneration. In many cases, although perhaps not in all, we must finally assume a congenital weakness of the affected nerve territory.

Such a defective nerve territory succumbs prematurely to the injurious influences which either are associated with the normal functions themselves or act upon them from without in some other fashion. At any rate, there is always a primary chronic degeneration of portions of the main motor-conducting tract, in one or another area and of varying extent. If we accustom ourselves to regard these three groups of symptoms as really identical, we shall be less puzzled by the slight variations which different cases may present than if we attempt to differentiate the disorders too nicely on account of unessential variations.

Diagnosis.—The diagnosis of a typical case of progressive bulbar paralysis has no difficulties if we only hold firmly to the definition of the disease and its symptoms as above depicted. Upon careful examination of the other muscles, and consideration of the course of the disease as a whole, we shall be able in each case to determine whether the bulbar trouble is the sole disease, or merely a part of a more extended degeneration of the motor tract. If there are no symptoms but those referable to the medulla oblongata, we must bear in mind that the phenomena of genuine progressive bulbar paralysis may be closely simulated by other bulbar diseases. The acute troubles, such as thrombosis or hemorrhage, although they produce similar symptoms, can easily be differentiated by the manner of their appearance, contrasting with the invariably slow development of genuine bulbar paralysis. It is, however, much more difficult to eliminate gradually forming tumors situated in the medulla oblongata or its vicinity. Here prolonged observation is frequently needed, until finally such phenomena appear as are foreign to typical bulbar paralysis. Such symptoms are disturbances of sensation and invasion of the upper division of the facial, the nerves of special sense, and the ocular muscles. The same is true of that rare trouble, diffuse sclerosis of the medulla oblongata.

It should also be mentioned that bilateral cerebral lesions may occasion so complete a paralysis of the tongue and lips, according to Lépine and others, as to simulate bulbar paralysis. Such cases have been termed "glosso-labio-pharyngeal paralysis of cerebral origin," or pseudo-bulbar paralysis. Indeed, in rare instances, a similar group of symptoms seems to be referable to unilateral cerebral disturbances. This is explained by assuming that the muscles involved upon both sides receive at least a portion of their motor nervous fibers from the same hemisphere. And yet in most of these cases of pseudo-bulbar paralysis the exclusion of the genuine disease is possible, because certain variations from the typical course of the disease are pronounced enough to set us right. Thus, there is an apoplectic onset or repeated apoplectiform attacks. The paralysis is not perfectly symmetrical. There is also paralysis of the extremities on one or both sides. The lips and tongue react normally to electricity without much atrophy, or there are marked signs of mental weakness. In most cases there is an extensive arteriosclerosis at the bottom of the whole morbid process, for the sclerosis of the cerebral arteries often leads to multiple foci of softening or to multiple hemorrhages.

Prognosis and Treatment.—Despite the unfavorable prognosis of progressive bulbar paralysis, we must at least try to check the progress of the disease. Electricity may perhaps be regarded as the most promising means to employ. To influence the seat of the trouble, galvanization is chiefly used. The poles are applied to the two mastoid processes, every day if possible, for two or three minutes, and the current is repeatedly reversed. We may also galvanize the sympathetic nerve and the affected muscles of the lips and tongue. Upon the muscles the faradic current may also be tried. When deglutition begins to be impaired, it is an excellent thing to excite the action of swallowing by galvanism. For this the anode is placed upon the nape of the neck, and the cathode upon one side of the larynx. At every cathodic closure (KaS), or every time that the cathode is passed across the side of the larynx, there is a reflex act of deglutition. The current should be of medium strength.

It may be well to prescribe further a resort to treatment by baths, or the "cold-water cure" may be cautiously tried, but we should not expect either of these procedures to have any special influence on the disease. The same internal remedies are recommended as in the chronic diseases of the cord, especially argentic nitrate, ergotin, strychnin, etc. For salivation, atropin may prove beneficial, in pills of $\frac{1}{120}$ gr. (gm. 0.0005), three or four to be taken daily.

The way of giving nourishment is important if deglutition is impaired. We should try carefully to avoid having the food go down the wrong way, lest pulmonary complications ensue. It is therefore wise not to defer the use of the stomach tube too long, through which we may introduce milk, eggs, wine, and the various infant foods.

In the distressing close of the disease, narcotics must be exhibited to lessen the patient's suffering, at least as far as we can.

CHAPTER II

THE RARER FORMS OF CHRONIC BULBAR PARALYSIS: PROGRESSIVE OPHTHALMOPLEGIA AND MYASTHENIA (ASTHENIC BULBAR PARALYSIS)

1. PROGRESSIVE OPHTHALMOPLEGIA

As we have seen, the form of chronic bulbar paralysis described in the previous chapter as "typical," is practically limited in its effects to the distribution of the hypoglossus, the labial division of the facial, and the pharyngeal muscles. Possibly the reason it extends no farther is merely that death is so speedy. But there are a few rare cases where the chronic degenerative process comes to involve other motor nuclei, together with the corresponding nerve fibers and muscles. Of course the clinical phenomena of these cases vary from the ordinary; and yet there is no real reason to distinguish them from common bulbar paralysis, particularly as all sorts of transitional forms are to be observed. Thus, we have ourselves noticed that there is sometimes a symmetrical and slowly progressive paresis of the upper division of the facial, and in particular of that portion which supplies the cheek, complicating the glosso-pharyngeal paralysis. In other cases we have seen the degeneration attack from the start the entire distribution of the facial, gradually producing a complete "*diplegia facialis.*" Sometimes, also, the ordinary symptoms of bulbar paralysis are accompanied by disturbances in the area of distribution of the ocular nerves, probably the result of degeneration of the corresponding nerve nuclei (see Fig. 197, page 437).

What seems very remarkable is that the process may sometimes be confined entirely to the ocular muscles. A. von Graefe named this condition progressive ophthalmoplegia. Another name is "anterior bulbar paralysis." In this form a bilateral paralysis of all the external ocular muscles progresses with extreme slowness and is perfectly symmetrical. The movements of the eye are therefore impaired in all directions. Diplopia is very rarely present. The pupil reacts to light, and almost always the power of accommodation is preserved. Finally, both eyeballs become absolutely motionless, and there is a well-marked though incomplete ptosis. There is, beyond a doubt, a progressive degeneration of the nuclei and fibers of the corresponding nerves—that is, the abducens and motor oculi; but with these the process may stop, spreading no farther. We have

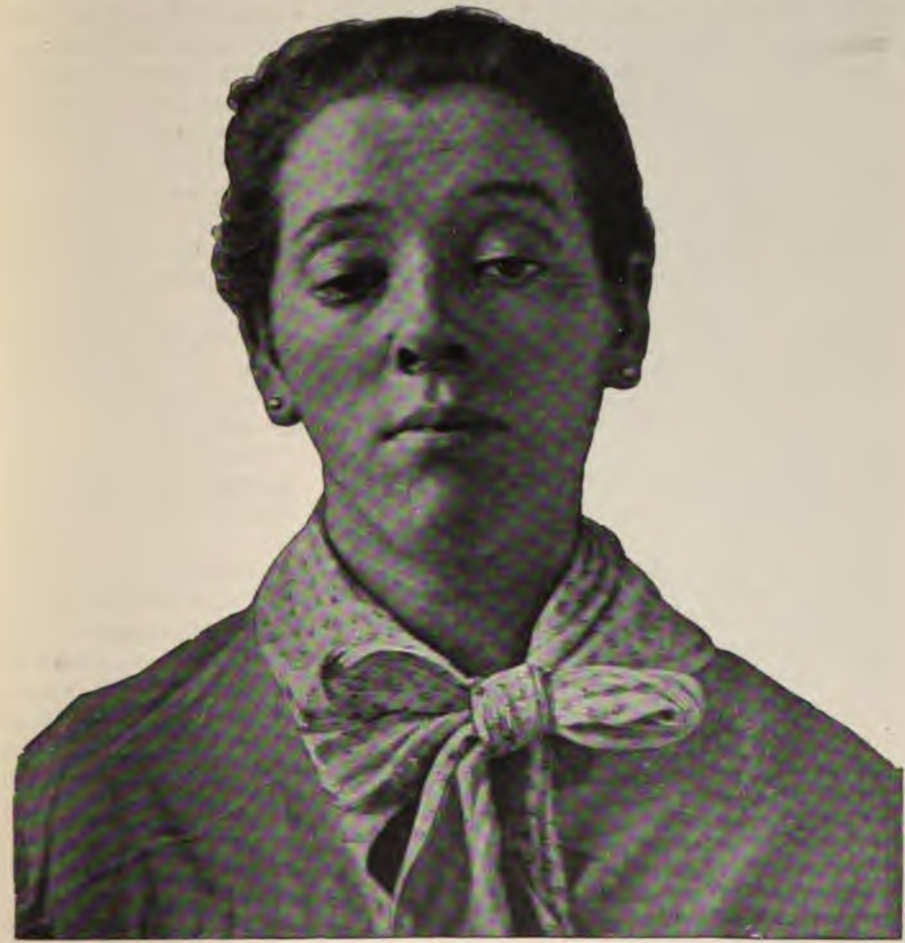

FIG. 198.—Progressive ophthalmoplegia. Paralysis of both eyeballs with bilateral ptosis. Weakness of both facial nerves. (Erlangen Medical Clinic.)

ourselves lately met with a patient who presented total bilateral ophthalmoplegia, in whom the condition had existed without the slightest change for fifteen years. Besides the oculomotor paralysis the muscles of the face (Fig. 198) and even those of the body are sometimes involved. On the other hand, in a few cases the process seems to be limited to only a part of the oculomotor nerves (as the abducens).

The diagnosis of genuine progressive ophthalmoplegia is usually easy, if we hold strictly to the definition of the disease as a degeneration of the motor nuclei and nerves exclusively (that is, completely analogous to progressive spinal muscular atrophy and bulbar paralysis). We shall thus avoid confusing it with those cases in which progressive ophthalmoplegia occurs as one symptom of a complicated morbid process of quite a different sort. Thus we must bear in mind particularly that tabes and general paralysis may lead to complete ophthalmoplegia. To these cases may be added those that follow syphilitic infection. Multiple sclerosis may also lead to almost complete ophthalmoplegia; and, finally, the same symptom-complex has been observed as a post-diphtheritic nervous disease.

The progressive ophthalmoplegias which develop after birth are also to be distinguished from the congenital defects in the movements of the eyes. Complete external ophthalmoplegia (that is, paralysis of all the external ocular muscles) has been repeatedly observed as a congenital condition, and also congenital bilateral abducens paralysis, congenital ptosis, etc. Autopsies of such cases are still lacking. There are probably defective conditions of the corresponding nerve nuclei, and perhaps of the nerves or muscles also.

2. PSEUDO-PARALYTIC MYASTHENIA. ASTHENIC BULBAR PARALYSIS

We will speak at this point of a very remarkable and peculiar type of disease, the milder forms of which were first described by Erb, and the severe and fatal forms by Oppenheim, Goldflam, and others. The symptoms of the affection consist of ptosis, paresis of the facial muscles, and disturbances of mastication, swallowing, and talking. The ptosis and weakness of the muscles of the cheeks and lips frequently lend a very characteristic appearance to the face (Fig. 199). On more careful observation it is seen that these symptoms are only in part uniform and persistent, but that they are due chiefly to an extremely rapid fatigue and exhaustion of the affected muscles (Goldflam). Such a patient can, for instance, utter a few sentences quite plainly, but, if he continues to talk, his speech grows more indistinct and inarticulate, and finally it becomes an incomprehensible lalling. The same thing is seen in chewing and swallowing: the first mouthfuls are chewed and swallowed in quite a normal fashion, but in a few minutes it becomes wholly impossible to take any more food. Besides these "bulbar" symptoms, which at first are the most striking, we also see very similar symptoms in the muscles of the extremities; usually the same rapid fatigue progressing to the most complete failure of their function. At first the patient can raise the arms without any particular difficulty, but after repeating this movement ten or twelve times the arm seems completely paralyzed, and recovers its power only after some time. One of my patients, if she had previously rested, could go up a flight of stairs very well, but on the second attempt she had to take hold of the banisters, and the third or fourth attempt failed utterly on account of the complete muscular weakness that ensued. As Jolly has found, we can detect this same abnormal fatigue by continued faradic stimulation of the muscles (so-called myasthenic reaction).

This clinical condition permits us to draw the conclusion that there is no permanent and complete atrophy or degeneration of the motor apparatus as a basis for the clinical symptoms; and, in fact, in those cases in which death finally ensued (in one or two years) from choking, insufficiency of respiration, etc., the anatomical findings in the medulla and the peripheral nerves have thus far been wholly negative (hence the term "bulbar paralysis without anatomical lesion"). Recently, since Weigert found a tumor of a persistent thymus gland with cellular metastases in the muscles in one case, particular attention has been paid to the anatomical condition of the muscles. Similar peculiar interstitial collections of cells in the muscles have also been repeatedly found in later cases; likewise a persistence of the thymus gland. It is not at all improbable, therefore, that some anomaly of the internal secretions is at the basis of this disease. However, the matter is not yet definitely settled. The insignificance of the anatomical lesions is the reason why marked improvement in this disease, and even complete recovery, can occur after several months, if the functional injury in the area of distribution of the affected nerves disappears. In order to express this distinction between the persistent paralyses due to coarse anatomical changes and the condition of mere pathological exhaustion now in question, I have proposed for the latter the name of "*asthenic paralysis.*" I designated the cases with predominating bulbar symptoms as "asthenic bulbar paralysis." We must, however, note that the disease is not limited exclusively to the territory of the bulbar nerves, although the bulbar disturbances are usually most prominent in the clinical picture. Cases may even occur where the abnormal fatigue and the consequent symptoms are found only in the extremities, without any implication of the bulbar

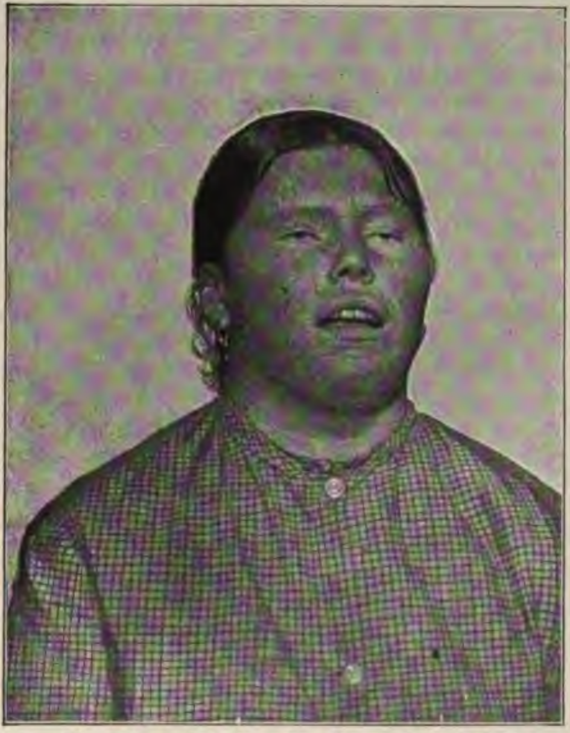

FIG. 199.—Bilateral ptosis and weakness of the facial nerve in myasthenia. (Personal observation.)

muscles. For this reason the name "myasthenia pseudo-paralytica," proposed by Jolly, is more appropriate and now generally employed.

From what has been said above, the prognosis is to be regarded as not absolutely unfavorable, but recurrences are to be dreaded even when recovery has apparently set in. Occasionally a fatal termination may suddenly occur. The treatment is first of all to provide the greatest physical rest and care for the patient. If it is difficult to take food, nourishment must be given cautiously by the œsophageal tube. We may try mild massage and galvanizations of the muscles, and central galvanization of the spinal cord and medulla oblongata. Internally we have given strychnin, arsenic, and phosphorus, without any perceptible benefit. On the hypothesis that the trouble was perhaps dependent upon some chemical toxic influence, the cautious use of sweating has also been recommended. The internal administration of thymus gland should also be tried.

[**Family Periodic Paralysis.**—Goldflam has described a peculiar affection, first carefully studied by Westphal, which merits brief mention. It consists of recurring attacks of paralysis, most marked in the legs, associated with loss of reflex and electrical excitability, without mental or sensory symptoms and with intervals of perfect health. Thirty-five out of fifty-three cases occurred in three families, but the families showed no special signs of nervous degeneracy. The attacks are apt to begin at puberty, and they are also apt to come on during sleep after some unusual muscular exertion. The frequency of the attacks varies greatly, from one or two in a lifetime to several times a week. The average duration of the individual attack is from ten to forty-eight hours. The attacks begin somewhat gradually, usually in the legs, and they may increase to complete paralysis of all the muscles of the limbs, body, and neck. The cranial nerves are rarely involved. The deep reflexes are abolished, and the plantar reflex is often absent. There is a quantitative diminution of electrical reactions, sometimes amounting to complete abolition, but there is no reaction of degeneration. The mechanical irritability of muscles and nerves is also lost. In two cases the heart became enlarged during the attack. The sphincters are not affected. The pathology is wholly unknown. Goldflam believes that the trouble is in some way connected with the muscular dystrophies. The prognosis is good so far as life is concerned, but the attacks usually recur through life. Treatment is unavailing.

Myotonia Congenita.—Oppenheim has recently described a rare condition occurring in very young children, and probably congenital, which is characterized by marked muscular atony and paralysis. This atony is extreme, and is most noticeable in the legs, but the arms and even the trunk may be affected. The tendon reflexes are absent or much diminished. The muscles are not much atrophied, but the electrical reactions are diminished or wholly lost. There is often a slight amount of motility preserved. The condition closely resembles poliomyelitis, except that there is no history of an acute onset and the atrophy is not as marked. In most cases there has been a gradual recovery. Oppenheim believes the condition due to an arrested development of the muscles, and Spiller has found this to be the case, and that the central nervous system and peripheral nerves were normal. Electricity seems to be of benefit in the treatment.—K.]

CHAPTER III

ACUTE AND APOPLECTIFORM BULBAR PARALYSIS

1. HEMORRHAGE INTO THE MEDULLA OBLONGATA AND THE PONS

HEMORRHAGE into the medulla oblongata and the pons is much more frequent than into the spinal cord, but it is much rarer than cerebral hemorrhage. As to its production, the same views are held as will be considered in detail under cerebral hemorrhage, in the next section. In the first place, there is probably always some disease of the blood vessels—that is, atheroma or miliary aneurism—and then some factor productive of increased arterial tension. There may be cardiac hypertrophy, nephritis, excessive bodily exertion, or alcoholism. Now and then injuries of the occiput are followed by an effusion into the medulla. It is not rare to have secondary and usually small ecchymoses in acute inflammation of the spinal cord (*vide infra*), and in purulent meningitis, or in connection with new growths which are richly vascular.

The anatomical conditions produced by bulbar hemorrhage are so completely analogous with those of cerebral hemorrhage that the reader may safely be referred to the succeeding section in regard to these also. The size of the lesion varies greatly. Bleeding extensive enough to affect the greater part of a transverse section is more frequent in the pons than in the medulla oblongata. If the blood is poured out close under the floor of the fourth ventricle, as has been repeatedly observed, it may break into the ventricle. If death be not speedy, the blood is mostly absorbed, and in its place develops either an "apoplectic scar" or an apoplectic cyst.

There may be slight prodromata, but the real symptoms of bulbar hemorrhage are very sudden. There is almost always a pronounced apoplectic seizure. The patient has a shock, falls down, and becomes dizzy or even unconscious. In other cases there may be headache, vomiting, tinnitus aurium, and clonic spasms, or even a typical epileptiform attack.

In the worst cases death is speedy, if not immediate. This is probably due in every instance to grave lesions of the respiratory and circulatory centers, rendering continued existence impossible. Sometimes the initial symptoms abate, whereupon the local results of the lesion become appreciable.

The first characteristic feature of bulbar paralyses now manifests itself, that disturbances are particularly great in the distribution of the bulbar nerves, while in cerebral apoplexy they never appear in the same way. Another point is, that these paralytic symptoms are combined with paralysis of the extremities in a peculiar way, as a result of the anatomical relations. For the same reason the distribution of the paralysis in the extremities may also be peculiar. Of the bulbar paralyses we may mention more or less complete paralysis of the tongue, and a consequent difficulty in articulation (anarthria); frequent inability to swallow; and paralysis in the distribution of the abducens, facial, and motor trigeminus, etc. If there is a lesion of the pyramidal tracts in the pons or medulla, we have paralysis of the extremities in addition to the specific bulbar symptoms. If the hemorrhage be extensive, all four extremities may be more or less completely paralyzed; but in most instances the paralysis is unilateral. In the larger number of hemorrhages into the pons there is crossed paralysis (hemiplegia alternans). The paralysis of the extremities is upon one side, and that of the facial is on the other side. This is a great aid to diagnosis. It is easy to see how this happens if we bear in mind that the cerebral fibers of the facial cross at a point much higher than the decussation of the pyramids, in which latter place the motor fibers of the extremities cross. Now a hemorrhage may be situated in one side of the pons, above the decussation of the pyramids but below that of the facial. This would occasion (*vide* Fig. 200, *y*) a paralysis of the facial on the same side with the lesion, and of the extremities upon the opposite side; but if the lesion be higher, above the place where the facial crosses over, all the paralytic symptoms would be on the opposite side of the body (*vide* Fig. 200, *x*).

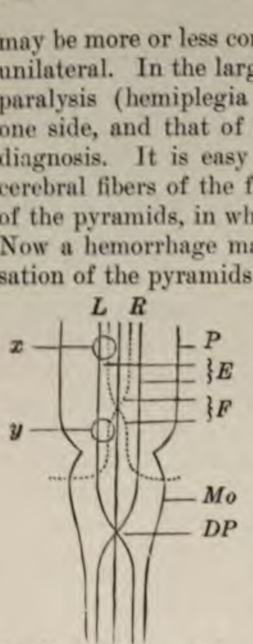

Fig. 200.—Diagram of focal diseases in the pons. *L*. Left. *R*. Right. *P*. Pons. *Mo*. Medulla oblongata. *DP*. Decussation of the pyramids. *E*. Fibers to the extremities. *F*. Facial fibers. *x*. Lesion in the upper half of the pons. *y*. Lesion in the lower half of the pons.

In other, rarer instances, we observe similar combinations; only some other bulbar nerve replaces the facial, such as the hypoglossus or abducens. Paralysis of associated movements of the eyes—i. e., a limitation of the combined movement of both eyeballs toward one side—is a very interesting symptom which is not infrequently observed in lesions of the pons. Many observations point to the fact that affections, particularly of the so-called posterior longitudinal bundle, produce a paralysis of associated movements toward the side of the lesion. Irritation of the posterior longitudinal fasciculus produces nystagmus or lateral deviation of the eyes. In a few cases the lesion is at the very decussation of the pyramids. This is extremely rare in hemorrhage, though somewhat more frequent in troubles of a different nature. The result may be that the motor fibers for one extremity are cut off before they cross, and those of the other extremity after they have crossed. Thus is produced the rare phenomenon of a crossed hemiplegia (hemiplegia cruciata)—i. e., paralysis of the arm on one side and of the leg on the other.

Sensory disturbances are frequently observed in lesions of the pons. They are due to lesions in the lemniscus (compare Figs. 114 and 116). The differences in the sensory disturbances between the posterior column type (disturbances of the sensations of touch, muscular and pressure sense) and the posterior horn type (disturbance of the pain and temperature sense), to which we have previously called attention, is also frequently very marked in lesions of the pons. The fibers for the sensations of pain and temperature appear to run mostly in the lateral portion of the lemniscus. The frequent involvement of the sensory tracts of the trigeminus, which produces sensory disturbances of the face, is characteristic.

A peculiar bulbar symptom-complex, which we have repeatedly seen, deserves special mention. It consists of a total paralysis of the muscles of deglutition, paralysis of the soft palate and vocal cord on the side of the lesion, as well as sympathetic paralysis (narrowing of the palpebral fissure and contracted pupil); on the opposite side of the body, a diminution of the pain, and especially of the temperature sense, on the trunk, arm, and leg, associated with so-called perverse temperature sense (i. e., sensation of warmth to cold stimuli). This peculiar symptom-complex is probably produced by lesions in the vicinity of the nucleus ambiguus and the adjoining substantia reticularis, the blood supply of which is probably derived from the posterior inferior cerebellar artery. [H. M. Thomas has noted, in occlusion of this artery, a sudden onset without loss of consciousness, but with intense vertigo, nausea, vomiting, and a tendency to fall to one side. There is usually pain or some sensory disturbance in the face on the side of the lesion, as well as sensory disturbance of the trunk and limbs on the opposite side—a crossed anæsthesia usually for pain and temperature alone, but sometimes for all qualities of sensation. There is a tendency to fall to the side of the lesion, and also incoördination of the limbs of the same side.—K.]

There are other symptoms, which are indeed rare, but which bear an important relation to certain nervous centers of the medulla. Thus, there may be marked respiratory disturbance; the pulse may become rapid or irregular; there may be vasomotor derangement, as shown by a rise of the cutaneous temperature and by a subjective sensation of warmth; and occasionally there are temporary albuminuria and glycosuria. The temperature of the body is generally normal at first, or nearly so; but in case of a fatal termination it often rises greatly, even to 107.5° F. (42° C.) or higher.

As to the prognosis of bulbar hemorrhage, speedy death has been repeatedly observed, as we have said. If the immediate effects be successfully withstood, the prospect becomes more favorable. The effusion is gradually absorbed, the symptoms of compression abate, and there is a steady progress toward comparatively good, or even perfect, health. More often, however, some of the paralytic symptoms remain stationary, either in the distribution of the bulbar nerves (the lingual or pharyngeal), or in the extremities, as shown by persistent hemiplegia. If this latter be the case, the subsequent contractures and other symptoms are the same as in ordinary cerebral hemiplegia.

The diagnosis of bulbar hemorrhage is based upon the apoplectic onset, and upon the presence of specific bulbar symptoms, such as disturbance of speech and of deglutition, and, most characteristic of all, if it occur, a crossed hemiplegia. The differential diagnosis between embolism and hemorrhage can hardly ever be made with certainty (*vide infra*).

The treatment, not only of the seizure but of the persistent paralysis, should conform to the principles which will hereafter be set forth in describing the treatment of cerebral hemorrhage. If the bulbar nerves present obstinate symptoms, we must employ the same means as in chronic bulbar paralysis, the most effective being electricity.

2. EMBOLISM AND THROMBOSIS OF THE BASILAR ARTERY

The medulla and pons receive their blood chiefly from branches of the anterior spinal, vertebral, and basilar arteries. These branches penetrate the anterior median fissure and then proceed to the nerve nuclei. A far smaller portion of the circulation flows through the "arteries of the roots." These are minute offshoots of the lateral branches of the basilar and vertebral arteries, which enter the cord at the roots of the nerves and penetrate to the corresponding nuclei. According to Duret, the nuclei of the hypoglossal and accessory nerves are supplied from the anterior spinal and vertebral arteries; those of the vagus, glosso-pharyngeal, and auditory nerves by branches of the upper end of the vertebral arteries; and the nuclei of the facial, trigeminus, and the three nerves to the ocular muscles by branches of the basilar. There may be individual exceptions to these rules. Occlusion by embolism or thrombosis of the arteries just named must occasion a secondary softening in corresponding portions of the medulla, and it is, therefore, a not very infrequent cause of apoplectic, or at least very rapidly developing, bulbar paralysis.

The causes of thrombosis or embolism in the arteries just mentioned are the same as we shall consider minutely when treating of cerebral softening. Emboli are most frequent in cardiac disease. They occur only in the vertebral arteries, oftenest in the left one, and are never primary in the basilar artery, but an embolus may be enlarged by thrombosis after lodging in one of the vertebral arteries, and then block up the basilar. Thrombosis is of more frequent occurrence, and results from chronic changes in the arteries, mainly atheroma or syphilitic endarteritis. The latter disease, one favorite locality for which is the basilar artery, is the commonest cause of acute softening of the pons.

The anatomical condition is likewise similar to that in cerebral softening (*q. v.*). In the region which is deprived of arterial blood by the occlusion of the affluent vessel, the acute anæmia entails necrosis and disintegration of tissue. A spot of "softening" results, made up mainly of vestiges of nervous tissue and numerous cells filled with granules of fat.

When the basilar artery is blocked up, the symptoms appear very suddenly. There is either an apoplectic seizure, or at least a very rapid development of paralysis (occupying only a few days). The symptoms of the first onset are, in all essential points, those of bulbar or even of cerebral apoplexy. Although there is usually no marked loss of consciousness in apoplectic bulbar paralysis, yet no great diagnostic significance can be assigned to its absence. The sudden obstruction of the basilar artery produces such a disturbance of the circulation even in anterior portions of the brain as may suspend consciousness. In some few instances this circulatory derangement may even give rise to choked disc, as seen by the ophthalmoscope. Often there are noticeable respiratory and cardiac symptoms, such as Cheyne-Stokes respiration, and rapid pulse.

If death be not immediate, and we are therefore enabled to make out the symptoms due to the local disturbance, we usually observe the same phenomena as have just been described under bulbar hemorrhage. There is sometimes paralysis of all the extremities, but usually there is trouble only upon one side. Then we have the characteristic crossed hemiplegia. The facial nerve or the nerves of the ocular muscles may be paralyzed. It has repeatedly happened that the paralysis seemed at first much greater upon one side, but after a few days changed over to the opposite one. This must be due to changes in the circulation; the thrombus grows larger, or a collateral circulation is developed. The specific bulbar symptoms are the familiar ones of all bulbar derangements —namely, lingual paralysis with resulting difficulty in articulation, pharyngeal paralysis, and rarely deafness, as a result of lesion of the acoustic center. Of course, the severity and extent of all these symptoms must vary according to the location and size of the spot of softening.

The prognosis of cases of this sort is almost always unfavorable. Death results in a few days at latest. It is often ushered in by a high temperature. Exceptionally, there is a transition into a chronic form.

We need say nothing about treatment, except that the same remedies are employed as in other acute bulbar diseases.

3. ACUTE INFLAMMATORY BULBAR PARALYSIS

(*Acute Bulbar Myelitis. Acute Pontine Encephalitis. Multiple Bulbar Neuritis*)

"Acute bulbar paralysis," in the stricter sense of the term, means a form of disease where marked symptoms of bulbar derangement (paralysis of deglutition, disturbances of speech, etc.) appear acutely—that is, within a few days or weeks. The anatomical cause of this clinical picture can be sought for only in an acute inflammatory disease of the medulla or the bulbar nerves. In a part of the cases, at least, we are probably dealing with a disease which is entirely analogous to acute poliomyelitis (*q. v.*). It is a rare disorder, and its ætiology is doubtful. There are usually mild prodromata—vertigo, headache, and, in one case of our own, painful sensations in the back of the neck. Evident bulbar symptoms very quickly follow. Usually the first of these is dysphagia. Not only is deglutition impaired, but the paresis of the soft palate and of the laryngeal muscles allows liquids to enter the nostrils or the larynx. The tongue also becomes gradually paralyzed, speech becomes indistinct, and, if the soft palate be involved, nasal.

Sometimes the extremities also become paretic, as a result of the extension of the disease to the region of the pyramids, but in many instances the extremities remain unaffected to the end. Paralyses of the facial nerve and of the ocular muscles are somewhat more frequent. Particular attention should be paid to the occurrence of conjugate paralysis of the ocular muscles ("paralysis of associated movements of the eyes"). This symptom always indicates a lesion of the pons. The temperature is sometimes a little elevated (100° to 102° F. [38° to 39° C.]), but not always. The pulse is almost invariably rapid; in one of my cases it was 148.

The course of the disease is apparently always unfavorable. Often death takes place in four to eight days, or it may be not till the end of two or three weeks. It is invariably preceded by all the tokens of paralysis of respiration.

As yet, few autopsies have been reported. Generally the medulla presents no macroscopic changes. Exceptionally, it can be seen to be softened and mottled with minute hemorrhages. The microscope detects abundant evidence of inflammation—granule cells, infiltration with nuclei around the blood vessels, thickening of the walls of some of the blood vessels, small, or even large, extravasations, swollen axis-cylinders, etc. It should also be borne in mind that precisely similar clinical phenomena seem often to be referable to peripheral changes, such as multiple neuritis affecting the bulbar nerves.

The treatment of acute bulbar paralysis is, of course, almost hopeless. In the early stages we should apply counterirritation to the nape of the neck, and we might prescribe mercurial inunction. It might be well to employ the constant current, applied at the back of the neck, and also in such a manner as to excite the movements of deglutition. We found injections of strychnin useless. Toward the end narcotics are indispensable.

CHAPTER IV

COMPRESSION OF THE MEDULLA

Acute compression and other injuries of the medulla are due most frequently to fracture or dislocation of the atlas and axis. As is well known, dislocation of the axis, or backward dislocation of the atlas, usually causes instant death.

Gradual compression is seen in chronic disease of the bones around the medulla, in caries and tumors of the occiput and of the first two vertebræ. Enchondroma of the base of the skull, new growths of the sphenoid at its junction with the occipital, tumors of the dura, and sometimes even tumors of the cerebellum—may all excite by their pressure the gravest bulbar symptoms. We should also mention aneurism of the vertebral artery at its upper end, and of the basilar, as capable of doing similar harm. In all these cases the main cause of disturbance is undoubtedly the mechanical pressure, either directly destroying the nervous tracts or interrupting the transmission of nervous influences, but the circumstances may be further complicated by hemorrhages, and sometimes perhaps by inflammation of the medulla itself.

The clinical phenomena of gradual bulbar compression resemble those of spinal compression in that they usually begin with symptoms of irritation in the distribution of those nerves the roots of which are first affected. There are neuralgia of the trigeminus, twitching of the facial muscles, tinnitus aurium, etc. If the compression becomes greater, there are more serious bulbar symptoms—disturbances of speech and deglutition, paralysis of the tongue, soft palate, face, and very likely motor (paretic or ataxic) and sensory symptoms in the extremities. Usually we also see general cerebral symptoms, such as vertigo, headache, vomiting, and sometimes epileptiform convulsions.

We cannot, of course, draw up a definite and rigid list of symptoms, since both the individual symptoms and the general course of the disease exhibit great variations according to the way in which the compression is brought about. The diagnosis can be made in those cases only where some ætiological factor such as trauma or caries of the vertebræ is known to exist. Aneurism of the vertebral artery is said by Möser sometimes to give rise to a loud systolic murmur heard between the mastoid process and the spine. In all other cases we can seldom do more than surmise the truth. Slow compression is distinguished from genuine progressive bulbar paralysis chiefly by the course it pursues, that is, there are initial symptoms of irritation—by the greater complexity of the clinical phenomena, such as sensory lesions and hemiplegia, and [illegible] by the asymmetry of certain symptoms. If the anterior part of the medulla is compressed in the region of the pyramids, there may for a time be no bulbar symptoms, but merely motor symptoms in the extremities. These [illegible] paretic or spastic.

The prognosis is almost always bad, as can be inferred from the nature of the causative disease. Death is brought on either by inhalation pneumonia, or by paralysis of respiration. Treatment must be purely symptomatic, and should follow the same rules as in progressive bulbar paralysis.

IV. THE DISEASES OF THE BRAIN

SECTION I

DISEASES OF THE CEREBRAL MENINGES

CHAPTER I

HEMATOMA OF THE DURA MATER

(Internal Hemorrhagic Pachymeningitis)

Ætiology and Pathology.—Hematoma of the dura mater is the name given to effusions of blood found on the inner surface of the dura mater. These effusions are of considerable area, but of moderate thickness, and are usually encapsulated. There has been much discussion as to their mode of origin, and views still differ. One is that the hemorrhage is the primary lesion, and that the connective-tissue membranes are developed only by the organization of the clot. This conception was originally the prevailing one, but was opposed by Virchow, who was led by the results of his own investigations to maintain that the hemorrhage was always secondary. The primary process he believed to be a peculiar sort of inflammation—" hemorrhagic pachymeningitis." This gave rise to a new growth of richly vascular connective tissue, into which the hemorrhage took place. Of late, however, the tendency is again to regard the hemorrhage as the initial change, at least in certain cases, and to refer it to an affection of the walls of the blood vessels which diminishes their power of resistance.

The mildest forms of internal pachymeningitis present anatomically a delicate membrane upon the inner surface of the dura mater, quite easily separable, of a reddish color, and dotted with numerous red and brownish spots. These spots are due to minute hemorrhages and collections of hematoidin. The membrane itself is a delicate interstitial tissue, traversed by numerous wide capillaries.

In more advanced cases the thickening is much greater. There are usually several layers, the newest and most superficial being nearest the brain. The oldest, which is in apposition with the dura mater, is composed of connective tissue that has already become rather firm and fibrous. It is evident from this lamellar structure that the whole process goes on by fits and starts. The clinical course of the disease will be seen from what follows to agree well with such a view. The effusions are sometimes very extensive, even larger than a hen's egg, and exercise no slight pressure upon the underlying cerebral parenchyma, The hemorrhage always takes place inside the mass, or between its layers. The effusion may, however, break through the innermost layer, so that the blood flows into the arachnoideal spaces; this is known as "intermeningeal apoplexy."

The favorite location of the hematoma is the parietal region. It is sometimes found at the base of the brain, in the posterior or middle fossa. Occasionally the hematoma is bilateral.

Hemorrhagic pachymeningitis is not a rare disease. It is sometimes found to exist in a moderate degree in chronic cardiac, renal, or pulmonary cases, which come to autopsy. Usually there have been no special symptoms, the lesion being discovered incidentally. It has been found in like manner in connection with a great many acute infectious diseases, such as typhoid fever and smallpox. It is a more important and more frequent complication in other chronic cerebral diseases; in particular, such as induce marked atrophy of the brain as a whole. It is especially common in general paralysis of the insane and in other forms of dementia. In the apparently primary hemorrhagic pachymeningitis chronic alcoholism is the commonest and most important causal factor. In hard drinkers it is not very unusual for the hematoma to be so extensive, if it occurs at all, as to cause grave cerebral derangement. Very likely changes in the vascular walls, such as atheroma and fatty degeneration, contribute an important part to the result in such patients. Injury is a second important cause. We have seen a case of hematoma of the dura with a fatal termination in a patient who some months before had received very severe blows on the head in a fight. Hematoma may also occur in all diseases where there is a general hemorrhagic diathesis. Thus it is seen in pernicious anæmia, leukæmia, and scurvy. Here certainly the hemorrhage is the primary event.

As might be inferred from the ætiological factors enumerated, the disease is found chiefly in advanced life, and much oftener in men than in women.

Symptoms.—Not infrequently a hematoma of the dura is found postmortem, which had during life been entirely unsuspected. Either the hemorrhage was not extensive enough to cause any symptoms, or the brain exercised that remarkable tolerance which it sometimes shows even when there are widereaching lesions; or such symptoms as may have existed escaped particular notice, in the severity of the more general symptoms (of typhoid fever or some similar disease). But in other cases hemorrhagic pachymeningitis excites grave symptoms, although they are seldom so characteristic as to reveal the diagnosis; for individual cases vary greatly, according to the size of the hemorrhages, their location, and the frequency of their recurrence.

The beginning of the disease is almost always rather sudden. It may even be like an apoplectic seizure. The symptoms are referable partly to the general effect of the hemorrhage upon the brain, and partly to the exact locality of the hemorrhage. The more general symptoms comprise headache, impairment of intelligence (that is, stupor or even complete coma), slow or irregular pulse, vomiting, and contracted pupils—all being symptoms of cerebral compression. Now and then we even find choked disc. In a few cases I have been particularly struck by the continued drowsiness. When aroused, the patients gave short but correct answers to questions, but almost immediately after relapsed into their peculiar somnolent condition.

Other phenomena are added to the above when the hematoma occupies its usual position, upon one side and in the neighborhood of the motor cortical region, or central convolutions. The hemiplegic symptoms are not infrequent, such as hemiparesis, and, from the irritation which the effusion produces in the motor centers, twitchings and convulsions in one half of the body. Sometimes these symptoms are limited to a single extremity or to the distribution of the facial nerve. Aphasia has been repeatedly observed when the hemorrhage was near the island of Reil. If the effusion increases, the motor disturbance becomes correspondingly aggravated, and it may become bilateral. Sensation is usually little impaired.

The further course of the disease varies greatly in different cases. In the worst cases there is speedy death, usually ushered in by deep coma. In others, the first symptoms are followed by improvement, although mild indications of cerebral pressure persist, such as headache or vertigo, or else local symptoms, such as hemiparesis. It is possible for the effused blood to be absorbed, and complete recovery to ensue; but usually new hemorrhages and corresponding symptoms arise. It is precisely this appearance of the symptoms in separate attacks, this frequent recurrence of severe cerebral disturbances, that is characteristic of hematoma of the dura mater. As already intimated, the way in which the anatomical lesions develop explains this perfectly. Thus, the disease may drag on for months and years, sometimes improving and sometimes getting worse again. Then some attack at last proves fatal. Arrest and actual improvement are still possible even in the later stages, although often the features of the case have meanwhile undergone essential alteration because of the progress of some causative disease. In general, the clinical phenomena of hematoma of the dura are often complicated and obscured by the coexistence of the primary disease.

Diagnosis.—The diagnosis is therefore difficult. The main points may be recapitulated as follows: First, the existence of ætiological factors, such as alcoholism, previous injury, or chronic cerebral disease; second, the sudden onset, and also the abrupt appearance of further symptoms, the alternation of rapid aggravation and improvement; third, the existence of symptoms of continuous increased intracranial pressure (headache, apathy, somnolence, etc.); and finally, fourth, the existence of symptoms which experience has taught us to refer mainly to the cortex of the brain—namely, unilateral convulsions, monoplegic paresis and contractures, and contracted pupils. Nevertheless, frequent errors in diagnosis cannot be avoided.

Treatment.—The question of the possibility of surgical intervention must be considered first (trephining and removal of the compressing blood clot). This has been done frequently with excellent results in cases of pure traumatic meningeal hemorrhage. But in hemorrhagic pachymeningitis proper, the conditions are very much more complicated, and therefore an operation is to be recommended only in cases which appear to be particularly suitable. In these cases it is very possible that relief of cerebral tension will be followed by good results. Any further treatment is purely symptomatic. In apoplectic shocks, ice to the head is useful; and if the patient be robust, it may also be advisable to use local depletion, by leeches on the temples or behind the ears. It is also customary to prescribe some such drug as senna or calomel for "intestinal derivation."

If the first onset is successfully withstood, the main points in the further treatment are general hygienic and dietetic directions, so as to guard as far as possible against fresh hemorrhages. Alcohol and excessive bodily or mental exertion should be forbidden. Of course, paralysis or other persistent disturbances may call for special attention.

CHAPTER II

PURULENT MENINGITIS

(Purulent Cerebral Leptomeningitis. Meningitis of the Convexity)

Ætiology.—Purulent inflammation of the dura mater has no clinical importance, for it is very rare, and occurs only as the result of the extension of disease from neighboring parts. We shall accordingly consider below purulent inflammation of the pia mater only. One important variety of this disease has already been discussed (see Vol. I, page 126 *et seq.*) as one of the infectious diseases, under the name of epidemic cerebro-spinal meningitis. There we saw also that the occasional sporadic cases of primary or "idiopathic" meningitis are probably identical ætiologically with those of epidemic meningitis. In all other instances, purulent meningitis is a secondary disease—that is, the specific pus coccus (usually the streptococcus pyogenes), which excites the purulent inflammation, originates in some other organ primarily, and affects the meninges only secondarily. We should, therefore, seek most carefully in every case of purulent meningitis, at the bedside and more particularly at the autopsy, to discover the way by which the pathogenic virus reached the meninges. We have no right to say that the case is one of primary meningitis, strictly so called, until we have made a most careful examination with a negative result. From a clinical standpoint, it is true that many cases of secondary meningitis do seem as if they were primary, because not infrequently the real primary disease excites insignificant symptoms, or perhaps no symptoms at all.

Secondary purulent meningitis is due most frequently to disease of the cranial bones, and in particular to disease of the petrous portion of the temporal bone, with the auditory apparatus therein contained. If we consider the anatomical relations of the middle and internal parts of the ear, we can easily understand why inflammation in them is not infrequently followed by meningitis. Usually it is caries of the petrous portion of the temporal bone, itself due to an otitis media, which leads to an irruption into the cranial cavity. This is especially apt to take place through the thin vault of the tympanic cavity. It may also extend from the mastoid cells, or by direct propagation along the sheaths of the acoustic or facial nerves, or along the vessels which lie in the petrosquamous suture. The dura is first attacked, and then the pia. In many instances the neighboring venous sinuses (transverse, cavernous, and superior petrosal) transmit the inflammation, being first attacked by a suppurative thrombophlebitis. Again, exceptionally, a purulent inflammation in the upper part of the nasal cavity or in the orbital cavity may lead to meningitis.

Another and frequent source of meningitis is found in the various injuries of the cranium. In the great majority of these cases there is an open wound, admitting infectious agents which are suspended in the air. The suppuration often commences in the spongy texture of the diploë, thence extending to the dura and pia, either directly or by way of a purulent thrombosis of some sinus into which the veins of the diploë enter. It was, indeed, often affirmed in the past that we could have a traumatic purulent meningitis without any open wound; but this can only possibly be explained, according to our present views as to the origin of purulent inflammations, by the assumption that the trauma furnishes a new point of attack for a suppurative infection which was previously present in the body. The statements are also to be entirely rejected that the heat of the sun's rays, striking upon an uncovered head, may excite purulent meningitis. In cases of fatal sunstroke we find marked hyperæmia of the meninges, but no inflammation.

Meningitis may have an intracranial origin; it is sometimes the sequel of cerebral abscess. No matter what starts the abscess, if it extends to the surface of the brain, it causes a more or less extensive purulent meningitis at that point. An abscess may burst into one of the lateral ventricles, and the infection be carried from that point to the pia at the base of the brain.

All the cases thus far contemplated allow of the explanation that the inflammation reaches the meninges by direct extension; but there is another group of cases where the agent that infects the pia mater originates at some distant part of the body, and is probably conveyed by the blood or lymph currents. These cases are often termed metastatic meningitis.

Of this sort is the secondary meningitis seen in connection with genuine lobar pneumonia, a combination already discussed (see Vol. I, page 262). The meningitis is also sometimes a complication of empyema, rarely of pyæmia and septicæmia, or ulcerative endocarditis, and very rarely of typhoid fever and the acute exanthemata (smallpox, scarlet fever), and of acute articular rheumatism. In each case we must, of course, determine whether the meningitis may not have a connecting link between itself and the primary disease, such as otitis in scarlatina, or secondary empyema in typhoid fever.

Pathology.—For the pathological anatomy of purulent meningitis we may refer mainly to the statements made in Vol. I, page 127, under Epidemic Meningitis, for the lesions are similar. The only way to determine whether a meningitis is secondary or primary is by finding or failing to find disease in neighboring or remote parts; for example, pneumonia. The seat of the meningitis will vary according to that of the primary inflammation, if there be any. If the meningitis is due to suppuration in the petrous bone or to an injury of the skull, the purulent exudation is usually most abundant in the immediate neighborhood of the primary lesion, between the pia and arachnoid. Thence it gradually extends along the surface of the brain, sometimes chiefly on the convexity and sometimes at the base. But in general it may be said that both the secondary and the metastatic varieties of meningitis, as a rule, affect the convexity, although this is by no means invariably the case. This rule explains why these cases are sometimes termed meningitis of the convexity, in contrast to tubercular meningitis, which latter, as we shall find, has a preference for the base of the brain, and hence is called basilar meningitis. The spinal pia mater is sometimes simultaneously attacked, but not so constantly as in primary or epidemic meningitis. The brain is almost always involved —the inflammation extends along the vessels which dip from the pia mater into the cerebral parenchyma. It is not a rare thing to find minute abscesses or ecchymoses in the interior of the brain. The whole parenchyma is usually moist, œdematous, and of a doughy consistency. The meningeal exudation exerts upon the brain a pressure which gives rise to important symptoms; by it the superficial cerebral convolutions are often considerably flattened. The lateral ventricles almost always contain more or less sero-purulent fluid.

Clinical History.—So varied are the primary diseases which may entail meningitis, that it is hardly possible to make a sketch of the disease which would suit all cases. If meningitis comes on during the course of pyæmia, pneumonia, or some other severe illness, its proper symptoms are often inextricably confused with those of the primary trouble; and when the skull or the brain has been mechanically injured, it is very hard to determine whether meningitis has been excited, because the trauma may of itself produce such serious effects. The following description, therefore, applies chiefly to cases of apparently primary meningitis, or to cases where the meningitis, although secondary, is well marked.

The beginning in such cases may be sudden, or it may be somewhat insidious. Sometimes the grave symptoms appear almost at once, accompanied by a chill and high fever. Sometimes there are for a time indefinite and more or less ambiguous prodromata, but almost always it is the headache which first attracts attention. This grows worse with more or less rapidity, and almost always becomes very violent. Exceptionally it may be insignificant. Not infrequently it varies considerably, being much worse at some hours or on some days than on others. The location of the pain is sometimes frontal, sometimes occipital, and sometimes over the whole head. Next in prominence to the headache, particularly in the later stages of the disease, is the mental disturbance. The patient complains of vertigo, becomes dull and stupid, or begins to wander. The delirium may be extremely violent, but usually there is depression rather than exaltation, and the stupor merges into coma. That the headache still continues may be inferred from the frequent raising of the hand to the head and the grimace of pain whenever the head is moved, until finally the coma becomes so profound that even these reflex actions cease.

Usually these general cerebral symptoms are attended by others referable to the particular locality affected. The neck is rigid. This is most marked when the posterior fossa and the medulla are affected. Then there are all sorts of paralytic or irritative symptoms in the distribution of the cranial nerves, due mainly to lesions of the nerves where they emerge from the base of the brain; there is derangement of the oculomotor nerves, as shown by paralysis or nystagmus; the pupils are unequal, or are contracted or dilated, and do not react to light; there is paresis of the facial, trismus, grinding of the teeth, etc. All these symptoms may be equally pronounced in other forms of meningitis. Sometimes we can detect optic neuritis with the ophthalmoscope. Other symptoms are due to cerebral disturbance, often apparently located chiefly in the cortex. Thus there may be twitchings of individual muscles, or even genuine convulsions in one or more limbs, or paralysis of one extremity or of half the body. Sometimes the autopsy explains these phenomena, but often we fail to find any marked anatomical lesion to correspond to them, and are obliged to ascribe them to circulatory or functional derangement. If the inflammation extend to the spinal pia mater, the entire back becomes rigid and painful. The patients can be raised up in bed only with difficulty. At every attempt to raise the trunk to a vertical position the legs are often involuntarily flexed and drawn up to the body (so-called Kernig's sign). [This sign may also be elicited by attempting to flex the extended leg on the trunk, which elicits pain and causes flexion of the lower leg on the thigh.—K.] Hyperæsthesia, muscular rigidity, disturbances of the reflexes, etc., are frequently found to exist in the legs.

Of the remaining symptoms, the fever is most important. Almost always the temperature is decidedly elevated, not infrequently reaching 104° or 105° F. (40° to 40.5° C.). The fever is, however, very irregular. There may be repeated chills with great elevations of temperature. The pulse is generally rapid, and often somewhat irregular. Exceptionally it is less frequent than normal, because of cerebral compression. Vomiting is not a rare symptom, particularly at first. There is almost invariably constipation, and the abdomen is often tense and concave. The urine is scanty, and often contains a trace of albumen. Secondary diseases are sometimes found postmortem, such as lobular pneumonia, due to inhalation of food during the comatose state.

The entire course of the disease occupies only a few days in very acute cases, and scarcely ever exceeds a week or ten days. The termination is almost sure to be fatal. In the few cases of recovery which have been reported the diagnosis is doubtful. In most instances deep coma precedes death, though sometimes it is ushered in by convulsions. There is often a great rise of temperature (107.5° F., 42° C., or higher) before the close of life.

Diagnosis.—The diagnosis of purulent meningitis is sometimes pretty evident; but it may be very obscure, so that we cannot always avoid confounding it with other severe acute diseases, such as typhoid fever, pyæmia, and general tuberculosis. The symptoms characteristic of meningitis, on which most stress should be laid in regard to diagnosis, are intense headache, rapid onset of grave cerebral disturbances, delirium and insensibility, stiffness of the neck, and disturbances in the distribution of the cranial nerves (especially impaired motion of the eyeball and optic neuritis). In many cases lumbar puncture (*vide* Vol. I, page 132) furnishes the most unequivocal data, if a purulent cloudy spinal fluid, or at least one containing many cells, is obtained in which pathogenic bacteria (streptococci, more rarely pneumococci) can be demonstrated by bacteriological examination. Great caution should be used in applying negative findings to diagnosis. In connection with these separate symptoms we must also always consider the whole course of the disease and any ætiological factors which may exist. Typhoid fever is excluded by its usually slower onset, the greater delay in the appearance of grave cerebral symptoms, the rose spots, the greater size of the spleen, the characteristic stools, and the peculiar fever curve. It may also be of importance in diagnosis that in typhoid fever there is usually no leucocytosis of the blood, while in purulent meningitis the number of leucocytes in the blood is increased. Severe septic and pyæmic diseases, including ulcerative endocarditis, likewise excite cerebral disturbances which might be misleading, but these diseases are to be recognized by their ætiology (external wounds, abortion, etc.), cutaneous ecchymoses, septic retinitis, swelling of the joints, and repeated rigors.

Uræmia may also simulate meningitis. Sometimes the character of the urine and the predominance of convulsions will set us right, but not always. We may state in conclusion that every one who sees many cases must (like ourselves) have repeatedly met with patients presenting symptoms of a severe and acute cerebral affection apparently primary, without demonstrable cause, and seeming to justify a diagnosis of meningitis, but yet yielding postmortem no signs of disease beyond "hyperæmia," "œdematous swelling," and similar changes of only secondary importance. We are as yet wholly unable to explain such cases. If marked meningeal symptoms occur in a patient and again disappear entirely, the possibility of a benign, milder variety of meningitis (so-called meningitis serosa) may be thought of. In this respect hysteria (*q. v.*) may also deceive us, since this affection occasionally appears under the guise of apparently marked meningeal clinical symptoms (hysterical pseudo-meningitis). The true psychical character of the disease is, however, generally soon recognized by careful unprejudiced observation.

Granting that meningitis exists, what variety is present? The ætiology is a great help in answering this question. We should endeavor to learn whether there has been traumatism or some old ear trouble. It is essential to employ the aural speculum. We cannot say that a patient has epidemic meningitis unless several cases occur simultaneously, although herpes is very characteristic, as it appears only exceptionally in other varieties. Usually tubercular meningitis also can be diagnosticated only by means of the ætiology. Its symptoms, of course, are in almost all particulars identical with those of purulent meningitis. In addition, the results of lumbar puncture, and occasionally also the ophthalmoscopic demonstration of tubercles in the choroid, decide the diagnosis. For further particulars see the next chapter.

Prognosis.—The prognosis of purulent meningitis is almost invariably wholly unfavorable, as follows from what has been said above. In the reported cases of recovery from meningitis the diagnosis must be determined with the greatest caution, but it is not impossible that sporadic cases of epidemic diplococcus meningitis may terminate favorably. We may also state that sometimes in severe infectious diseases "meningitic" symptoms may appear which lead us to fear the worst, but which finally disappear. In acute purulent inflammation of the middle ear, especially in the young, meningitic symptoms (headache, stupor) sometimes set in, which also seem threatening, but which disappear. It is, of course, usually impossible to determine positively the anatomical processes in such cases.

Treatment.—The treatment of the different forms of meningitis varies but little. Locally, the favorite remedies are ice applied to the head, which should be shaved if practicable, and local depletion by means of leeches behind the ears or on the temples. Many physicians recommend cutting off the hair and rubbing in antimonial ointment, or applying ethereal tincture of iodin. We have never tried this. Cool baths with douching cannot be employed unless the patient can be moved without too much pain. For violent pain or great restlessness we must use narcotics. The best is morphin subcutaneously. We cannot hope for much benefit from other internal remedies, such as iodid of potassium or calomel. The therapeutic value of lumbar puncture, which is undeniably of the greatest diagnostic value, is doubtful. Nevertheless, after the evacuation of large quantities of cerebro-spinal fluid we occasionally see a remission of the cerebral pressure symptoms, which, however, is generally only transitory.

Prophylaxis demands, above all, prompt recourse to the otologist for aural trouble of any kind, and strictly aseptic treatment of all injuries of the skull. Even in cases of meningitis that have already begun, surgical interference (trephining the mastoid, etc.) may perhaps often check the advance of the inflammation and lead to recovery.

CHAPTER III

TUBERCULOUS MENINGITIS

(Basilar Meningitis)

Ætiology.—Tuberculosis of the leptomeninges is always a secondary affection—a sequel to previously existing tuberculous disease of some other organ. Why the pia mater should be so often singled out for secondary infection with the tuberculous virus, or what path that virus traverses to reach the pia, we can hardly say. We can merely state what the other tubercular diseases are, which, as experience shows, entail tuberculous meningitis most frequently. These primary affections may be of themselves productive of grave clinical phenomena, the meningitis merely adding to the complexity of the picture. Again, the primary trouble may not have betrayed itself at all, or its symptoms may have been long ago arrested, so that the meningitis seems to be a primary disease. In some cases even the most careful examination will fail to detect the origin of the trouble.

Tuberculous meningitis is oftenest a sequel to pulmonary tuberculosis. It may appear as a terminal complication in cases of advanced phthisis, or it may come on while the signs of pulmonary disease are as yet very slight. Next in order as a causative affection comes tuberculous pleurisy. This origin is not infrequent. As we have already seen, most cases of apparently primary pleurisy are due to tubercle. This statement is supported by the fact that it is not very exceptional for the symptoms of tuberculous meningitis to supervene suddenly upon what had seemed to be genuine convalescence from pleurisy. In children, and sometimes in adults, the virus may be carried to the meninges from cheesy, tuberculous, bronchial, or mesenteric glands, or from tuberculous or "fungous" disease of the bones or joints. Another danger to adults is tuberculous disease of the genito-urinary apparatus. It should also be noticed that a single large tubercle in the brain may lead to miliary tuberculosis of the meninges. In short, we see that it is not impossible for any tubercular infiltration, wherever situated, to communicate infection either to the meninges alone (in some remarkable way), or simultaneously to them and many other organs. In this latter case, where in all probability the blood carries the virus through the system, the meningitis is merely a part of a general miliary tuberculosis (see Vol. I, page 320), while in the former case the reason why the meninges are exclusively or primarily affected depends upon the special form of the infection; but we are almost wholly ignorant of the way by which the tubercle bacilli reach the meninges. We are ourselves disposed to suspect that, in many cases at least, the infectious material first enters the arachnoidal sac of the spinal cord through the lymph sheaths of the nerves (e. g., the intercostal nerves), and thence extends upward to the base of the brain.

We sometimes hear the attack ascribed to such causes as overexertion, mental excitement, traumatism, etc.; but we need hardly say that these cannot be properly regarded as ætiological factors, and that usually they are merely coincidences. Age, however, does have an influence; children are much oftener attacked than adults, although the latter also are liable to the disease.

Pathology.—As in tuberculosis of serous membranes, so in tuberculosis of the pia, there are two effects of infection to be distinguished from each other: (1) the development of the specific new growth—that is, of miliary tubercles; and (2) the inflammation. The relative degree of these two varies. Sometimes the tubercles are very abundant and the inflammatory exudation comparatively scanty; and in other cases the inflammation is considerable, although relatively few tubercles are discoverable. The tubercles are usually found in greatest number along the course of the larger blood vessels, and therefore chiefly in the furrows and clefts of the surface of the brain, in the fissure of Sylvius, at the chiasma, the pons, the medulla, and the cerebellum. In general, the base of the brain is usually more affected than the convexity—hence, as we have said, the name of "basilar meningitis." There are, however, exceptions to this rule. We very often find that the region supplied by one or more arteries suffers above other parts; this must be due to the manner of infection. The inflammatory lesions consist of hyperæmia, usually well marked, and a sero-gelatinous exudation of variable amount. That this exudation is partly cellular can always be proved by the microscope, and often even macroscopically from the great cloudiness of the pia; but still we seldom find enough to justify us in calling the process one of genuine purulent inflammation. Small hemorrhages into the pia are quite often found. The brain itself is usually flattened from the pressure of the meningeal exudation. Often the inflammation involves the brain substance itself, as shown microscopically by tubercles, inflammatory changes, and capillary hemorrhages. The ventricles usually, although not invariably, contain a hydrocephalic effusion. This led earlier observers to term the disease "acute hydrocephalus." The effusion is serous, but generally turbid from cellular constituents, and not infrequently tinged with blood. The choroid plexus is engorged, and it may present tubercles. The spinal cord, in the majority of cases, shares in the tuberculous disease. Here, too, we find inflammation of the pia and miliary tubercles.

Clinical History.—Tuberculous meningitis almost always begins with a prodromal stage, which is often brief, but which may last one or two weeks, or even longer. The patient may be apparently well (*vide supra*) until this comes on, or he may have already shown signs of some other tuberculous affection. He now feels somewhat ill at any rate, and begins to complain of headache, worse at some times than at others. There is anorexia and very often constipation. Another frequent prodrome is an attack of vomiting, which may or may not recur. Sleep is disturbed, either by the headache or by a certain general restlessness. We have occasionally met with cases where the illness began with pronounced mental disturbance. The patient became irrational and said and did queer things, and then a few days later there appeared distinctive meningeal symptoms. In two patients, who were hard drinkers, the disease began just like delirium tremens.

After an initial period of variable duration, the general health becomes more and more impaired. The headache increases. The patient takes to his bed, begins to be delirious, and soon presents well-marked symptoms of grave brain trouble. Intelligence becomes more and more impaired. The patient is sleepy, and he can be roused imperfectly by the voice, if at all. At the same time he is usually quite restless at first, grasping at invisible objects in the air, picking the bedclothes, and continually moving his legs. The delirium may be low or noisy; the patient may keep up a constant singing, screaming, or whistling. The persistence of the headache even in this stage is shown by the facial contortions and complaints of the sufferer, whenever there is a temporary approach to consciousness. There is also, as a rule, decided tenderness in the nape of the neck on pressure, frequently accompanied by great stiffness of the neck. Sometimes there is stiffness of the entire spinal column, and pain in the same. This is certainly due to the coincident spinal meningitis. Kernig's sign (*vide supra,* page 457) is also not infrequently observed.

Another group of symptoms in the distribution of the cranial nerves are identical with those seen in other forms of meningitis. Ptosis is not infrequent, on one or both sides, due to paresis of the motor oculi. There is strabismus, either internal or external. Symptoms of irritation of the nerves governing the movements of the eye are very frequent, especially in the early stages of the disease. Thus, we see slow involuntary lateral movements of the eyeballs, and sometimes nystagmus. The pupils are often unequal; they may be enlarged or contracted, and often they undergo marked and repeated variations in size. The reaction of the pupils to light is usually sluggish, and it may be absent. With the ophthalmoscope we find not infrequently neuritis, or choked disc. In some instances, but not in all, we find also tubercles in the choroid, which, of course, greatly assists diagnosis. Sometimes there is occasional twitching in the distribution of the facial nerve, or a slight tonic contraction, or again paresis on one side. The natural explanation of all these phenomena is that the nerve trunks are interfered with at the base of the brain, either by the pressure of the exudation or by participation in the inflammatory process, or by the minute hemorrhages which sometimes take place into the sheath of the nerves.

Disturbances in the extremities may be caused by various lesions. Symptoms of motor irritation are apparently referable for the most part to changes in the cortex of the brain. We see occasional twitching of larger or smaller groups of muscles, or rarely convulsions. These latter may be unilateral, or limited to a single extremity. Sometimes there is well-marked paresis of one half the body or paralysis of one limb, or there may be aphasia, although it is only in a part of the cases that we find postmortem any lesion which explains these symptoms. In most cases, however, there is a particularly large collection of tubercles in certain places upon the cortex cerebri, occasioning a local compression or an inflammatory œdema, which in its turn excites the phenomena mentioned. Sometimes the brain substance itself is found in a state of red softening underneath these spots. Another not very rare symptom is a peculiar stiffness of the limbs, due either to direct irritation or to reflex action. The reflexes in the lower extremities are generally exaggerated at first, but later on become diminished, and finally abolished. The reflexes upon one side may be more vigorous than upon the other. As to sensation, it is hard to reach definite conclusions, because of the patient's stupor. Sometimes there is well-marked cutaneous hyperæsthesia, probably referable to an implication of the spinal cord in the process.

The behavior of the pulse and temperature is interesting. The temperature is usually elevated, but often only to a slight extent—that is, varying between 100.5° and 102° F. (38° and 39° C.). Often the temperature falls quite low, only to rise again, the alternations being at irregular intervals. Exceptionally the temperature may remain high (104° F., 40° C.) most of the time. Toward the end there is usually a decided change in temperature, either upward or downward. In many instances there is a very low temperature before death; in two cases we have seen a temperature of 88° F. (31° C.). Or the temperature may rise to 106° F. (41° C.) or higher just before death. The pulse is often abnormally slow in the early stages of the disease, even numbering only 40 to 50 beats per minute. This is referable without doubt to the increased intracranial pressure. Later on the pulse becomes small and rapid. The transition may be very sudden. The vagus is at first irritated, and then paralyzed. The pulse is often irregular.

Respiration is generally moderately accelerated. If the breathing is very deep and rapid, we should always think of simultaneous miliary tuberculosis of the lungs. Toward the close of the disease the respiration often assumes the Cheyne-Stokes type: there is a long pause, followed by very superficial and gentle respiration, which gradually grows deeper and deeper, then diminishes again, and is succeeded by another complete pause. This symptom is always most ominous, for it indicates that the excitability of the respiratory center is already greatly impaired.

Symptoms referable to still other organs are few. Vomiting is rare in the later stages of the disease. The abdomen often presents a "boat-shaped" concavity, as the result of tonic muscular contraction, and is hard and tense. There is almost always constipation. The spleen may be somewhat enlarged. The urine sometimes contains a trace of albumen. On account of the drowsiness, it is usually voided in the bed or retained in the bladder. Almost invariably there is rapid marasmus.

The entire duration of tuberculous meningitis varies somewhat, chiefly because of the varied length of the first stage. When the disease is once fully developed, the illness seldom lasts more than three to ten days longer. Frequently the illness is divided into three stages: (1) The stage of cerebral irritation, with headache, stiff neck, vomiting, and delirium; (2) the stage of cerebral compression, due chiefly to the hydrocephalus, and causing drowsiness, slowness of the pulse, paralysis of the oculomotor nerves, hemiplegia, etc.; and (3) the paralytic stage, presenting deep coma, relaxation of the previously contracted muscles, accelerated pulse, and marked variations of temperature. Such a division is too schematic to correspond accurately to the real phenomena, but it will, nevertheless, often aid us in getting a general idea of the course of the disease.

The termination of tuberculous meningitis seems to be inevitably fatal. Sooner or later the patient loses consciousness completely, his pulse grows very small and rapid, his respirations irregular and intermittent (Cheyne-Stokes), his temperature, as we have said, either rises high or falls far below normal, and, finally, death is ushered in by paralysis of all the vital functions. A few physicians have reported cases of recovery, but was the diagnosis correct? While we would by no means absolutely deny that recovery from tubercular meningitis may occur, it would certainly be very difficult to prove, in any particular instance, that such a thing had happened.

Tubercular Meningitis in Children.—The disease is so prone to attack children that it seems desirable to subjoin a few remarks about the peculiarities of the affection as observed in them.

Often the little patient is pale and weakly, with tuberculous antecedents, but sometimes apparently healthy and vigorous children are attacked. Tuberculous meningitis may be the sequel of measles, whooping cough, or some other disease, which has occasioned the development of the tubercular process. Usually the severer symptoms are preceded by a rather long prodromal stage (a week or ten days), which often does not forebode the severity of the disease that is beginning. The child looks pale, has a poor appetite, grows thin, is restless in his sleep, is out of humor and cries during the day, is often tired and sleepy, does not play naturally, the tongue is coated and the bowels constipated. The second stage is generally ushered in by headache and vomiting. The headache is not very often violent, but children complain with remarkable frequency of pain in the abdomen and chest. The cause of this symptom is unknown. The pulse is almost invariably slow, often somewhat irregular and whirring (that is, as if it trembled beneath the finger), and it frequently undergoes surprisingly rapid changes in rate—for example, varying twenty or more beats inside of a few hours. Very early the child becomes dull and drowsy. Frequently it emits a peculiar deep sigh, or that sudden loud scream or "cephalic cry" which physicians long ago learned to recognize and fear. The symptoms referable to the cranial nerves and the nervous disturbances in the limbs are similar to those seen in adults. Strabismus is almost constant. Very often there are trismus and a distinctly audible grinding of the teeth, most distressing to the bystanders. Trousseau laid weight upon the appearance of red spots (*taches cérébrales*) upon the skin after it has been mechanically irritated, but these have no diagnostic value. They are due to increased reflex action, and are seen in all sorts of acute diseases. The fever is irregular throughout and generally, as in adults, not very high, 100° to 102° F. (38° to 39° C.). Respiration is usually rapid, and often is irregular.

The change from bad to worse is almost always announced by a rapid increase in the pulse rate, to 160 or 200. The child becomes completely comatose. Very often there are repeated epileptiform convulsions, either universal or affecting single extremities. Death is usually preceded by a decided rise in temperature.

Diagnosis.—When the symptoms are pronounced, the diagnosis of meningitis is easy, and we have merely to determine just what variety is before us. That the disease is due to tuberculosis is never to be ascertained by means of the meningeal symptoms themselves, but is rendered possible only by the ætiology, if that be discoverable.

In this respect the examination of the cerebro-spinal fluid obtained by lumbar puncture is of the greatest importance. In addition to a cellular turbidity of the fluid, the demonstration of tubercle bacilli, which is possible in about one half of the cases, above all, decides the diagnosis. The centrifugalized sediment, or the coagula which are formed in the fluid, are examined by the ordinary staining methods. A negative result, of course, does not militate against tuberculous meningitis. The examination of the sputum for tubercle bacilli must also not be omitted in every suspicious case, even when there are no physical signs of pulmonary disease. In addition to the examination for bacilli, the general conditions, particularly (1) heredity and (2) the evidence of previous or existing tuberculous affections in other parts of the body, must be considered here as in all tuberculous disease. Under this second head are to be considered scrofula, diseases of the bones and joints, pulmonary tuberculosis, pleurisy, and tuberculosis of the genitals or of the choroid. If our search be unsuccessful, we may sometimes get a hint of the truth from the general appearance of the patient; for example, he may be pale, or narrow-chested. And, again, the absence of trauma, aural disease, or epidemic meningitis will make tuberculosis more probable.

In its early stages, or when it varies from the usual course, tuberculous meningitis may be very difficult to diagnosticate. This is particularly true when the patient is a child. The early malaise and vomiting are treated as "ordinary gastric catarrh" until the grave cerebral symptoms disclose the mistake in diagnosis. In such cases we should be careful not to disregard the initial slowness and irregularity of the pulse. This alone should make our prognosis guarded. The fever may be prominent at the commencement, and tempt us to call the case one of incipient typhoid fever; and, indeed, the correct diagnosis is often impossible until the disease develops further. In regard to this, see the preceding chapter on purulent meningitis, where the exclusion of severe septic diseases and of uræmia is also considered.

Before the autopsy we must remain in great uncertainty as to the number and distribution of the tubercles, the existence of a large effusion into the lateral ventricles, etc. We are often amazed at the post-mortem examination by the apparent insignificance of the lesions. Paralysis of the cranial nerves (eyes, face) implies that the base of the brain is gravely affected. If such symptoms are absent, and there are mental disturbances, and symptoms of motor irritation displayed in the extremities, we are led to infer meningitis of the convexity. If the nervous disorder be mainly unilateral, we may assume that one hemisphere is more affected than the other. Many cases of circumscribed tuberculous meningitis produce a clinical picture which resembles that of brain tumor. Cases of this class cannot be sharply differentiated from cases of so-called solitary tubercle.

Treatment.—However hopeless the prospect, we are nevertheless bound to employ all the remedies at our command, as in other forms of meningitis. Above all, we should be thorough in applying ice to the head, and we may also try inunctions of iodoform ointment to the shaven scalp, and lukewarm baths, with douching. The inunction of mercurial ointment (especially on the head, neck, and back) has also been recommended. The most common internal remedies are calomel—gr. ss. to j (gm. 0.03 to 0.05) for a child every two hours—and infusion of senna. Iodid of potassium may also be freely given, gr. xv (gm. 1) daily to a child, and two or three times as much to an adult. Whether it does any good is extremely doubtful. If the patient is very restless, narcotics are indispensable. Stimulants are often given in the last stage of the disease, but generally without effect.

In regard to prophylaxis, we need merely refer to the general statements (Vol. I, page 309) as to prophylaxis from the various tuberculous diseases.

CHAPTER IV

THROMBOSIS OF THE CEREBRAL SINUSES

Ætiology and Pathology.—The sinuses of the dura mater sometimes present a thrombosis, under circumstances similar to those which induce the same process in other veins. The most frequent occasion for such thrombosis is marasmus, however brought about, with the accompanying feebleness of circulation. This is the explanation of those not very rare cases found among wretched and ill-nourished children in the first year of life, and also in patients with phthisis, cancer, etc. We must note in particular that in severe anæmic conditions (especially in chlorosis) sinus thrombosis is comparatively common.

Halfway between marantic thrombosis and the inflammatory variety immediately to be described come those cases which are seen in typhoid fever and other severe acute infectious diseases. Here the specific virus apparently plays an important part (just as in thrombosis of the femoral vein), although very likely the cardiac weakness also promotes the thrombosis.

Genuine inflammatory thrombosis (thrombophlebitis)—that is, thrombosis in connection with real phlebitis of the sinus—is almost always due to the extension of inflammation from some neighboring part. The most fruitful cause is suppuration in the petrous portion of the temporal bone, the result of otitis or caries. This spreads to the walls of the transverse or petrosal sinuses, which are close by. Also wounds, necrosis, or other affections of other cranial bones may excite thrombosis; likewise, although seldom, deep-seated inflammation of the soft parts of the head and face, large furuncles or erysipelatous abscesses, may produce the same result. The reason why sinus thrombosis may arise from the causes last mentioned lies in the fact that the external veins of the skull are also connected with the sinuses of the dura mater by means of the emissary veins of Santorini. It is easy to understand that purulent thrombophlebitis of the cerebral sinuses is often found associated with purulent meningitis or brain abscess.

Thrombosis due to marasmus is most frequently found in the superior longitudinal and transverse sinuses, while the inflammatory variety usually occupies either the transverse, petrosal, or cavernous sinuses. Of course the thrombus may grow out from its original sinus into neighboring ones. Important clinical symptoms are caused by secondary venous stasis in the veins which empty into the occluded sinus. These symptoms are most pronounced when the longitudinal sinus is affected; objectively, we find the meningeal veins which lie on the surface of the brain distended and tortuous; and often there are extensive meningeal ecchymoses. Even the cerebral parenchyma beneath shows distinct evidence of passive congestion, and minute capillary hemorrhages have been repeatedly found in it.

Symptoms.—In some instances moderate thrombosis of the cerebral sinuses has been found postmortem, although there had been no symptom suggesting it before death. In other cases the thrombosis does excite undeniable cerebral disorder, but the symptoms are so general and ambiguous that the most we can do is to suspect the existence of the clot without being at all certain about it. Sinus thrombosis in marantic children usually causes coma, stiffness of the neck and back, strabismus, nystagmus, and sometimes clonic spasms in the face and limbs. The symptoms in adults are similar, comprising headache, drowsiness, occasionally delirium, sometimes coma, and varying symptoms of irritation or of paralysis in the distribution of the cranial nerves (nystagmus, strabismus, trismus) and in the extremities. But even all these symptoms are insufficient to make the diagnosis certain. They must be reënforced by certain other phenomena more distinctly referable to the peculiar circulatory disturbances occasioned by the thrombosis. Occlusion of the cavernous sinus sometimes excites well-marked symptoms of stasis in the ophthalmic veins. Thus, the retina may be seen through the ophthalmoscope to be passively congested, there is œdema of the eyelids and the conjunctiva, the eyeball is unusually prominent, and the frontal vein is distended. In case of an inflammatory thrombosis, the periphlebitic swelling may cause symptoms in the distribution of the neighboring nerves, especially paresis of the oculomotor or abducens, or trigeminal neuralgia. In thrombosis of the transverse sinus an œdematous swelling is occasionally seen behind the ear, near the mastoid process. If the clot projects into the petrosal sinus or even actually into the internal jugular, the lower part of this vein collapses. And inasmuch as the external jugular can empty itself with unusual ease into the unfilled internal jugular vein, the external jugular is also affected and becomes less prominent upon the affected, than upon the normal, side. It is to be noted, however, that this condition of unequal distention of the jugular veins is only rarely marked. Sometimes it is even possible to feel the thrombus in the internal jugular. Such thrombosis causes pain and swelling in the neck on the affected side. When the superior longitudinal sinus is blocked up, there are symptoms of nasal engorgement (epistaxis), and distention of the veins about the temples, which veins are connected with the longitudinal sinus by emissary veins. We must confess, however, that all these special symptoms are comparatively rare, and often difficult of demonstration even when present.

Primary thrombosis of the transverse sinus with extension of the clot into the venæ magnæ Galeni, thus causing internal hydrocephalus, has been repeatedly observed in the chlorotic. The clinical picture consists of the rapid onset of severe cerebral symptoms (headache, vomiting, somnolence), which rapidly progress to deep sopor and lead to death in a few days. When unilateral paralysis and symptoms of motor irritation occur (choreic twitchings, epileptiform attacks), they are due in part to hemorrhages from stasis in the brain substance.

The symptoms become more complex where there is a suppurative phlebitis, because there are usually pyæmic symptoms as the disease progresses. Thus, there may be rigors and high fever, pulmonary abscess, suppurative arthritis, etc. We have already mentioned the combination of thrombosis of a sinus with purulent meningitis or brain abscess.

Prognosis.—The prognosis is almost always bad, both because of the nature of the causative disease and because of the grave cerebral derangement or the secondary pyæmia. Spontaneous recovery may occur, but it is rare. The nervous symptoms may last at times only a week or two, but they may extend over several months.

Treatment.—Treatment in most cases must be purely symptomatic. Only when a purulent sinus thrombosis follows ear disease can surgical interference give very satisfactory results. The details of such treatment are to be found in the text-books of operative aural surgery. Compare also the chapter on Brain Abscess.

SECTION II

DISEASES OF THE BRAIN SUBSTANCE

CHAPTER I

DISTURBANCES OF CIRCULATION IN THE BRAIN

(Cerebral Hyperæmia. Cerebral Anæmia)

It is presumable that so sensitive an organ as the brain is much influenced even by slight disturbances of circulation; but as yet we have little knowledge of the production and character of such disturbances, their existence being very difficult to demonstrate. There are many instances where marked cerebral symptoms justify the assumption that the brain is in some abnormal condition, where yet there are many arguments against any marked anatomical lesion. Here we surmise that there is some circulatory derangement, although we have no actual and direct arguments to rely upon. For example, we refer to this cause certain cases of headache, of sensations of pressure in the head, of vertigo, of general hyperæsthesia, and of that protean and nevertheless easily distinguishable disease known as cerebral neurasthenia (*q. v.*). At present, however, we cannot determine how far circulatory disturbances are actually concerned in these cases, or of what kind they are, or whether there may not be purely functional disease of the brain entirely independent of changes in the blood vessels.

Certain groups of cerebral symptoms, which come on in paroxysms, seem the most clearly referable of all to circulatory disturbance. There can hardly be a doubt that the phenomenon known as fainting or syncope is due to sudden cerebral anæmia. As is well known, fainting is usually the result of a clearly demonstrable cause. Frequent and familiar causes are emotional excitement, terror, unusual psychical impressions (like the sight of blood), the influence of great heat, or great physical strain, as by long standing. The condition of the stomach has certainly a great influence in many cases. There are many persons who, if they go long beyond the usual time without eating (particularly if they go without breakfast), are very liable to syncope. Some individuals are especially subject to fainting fits. Such persons are often slight and anæmic (for example, convalescents), but some are in appearance robust and vigorous. Many children are subject to fainting.

The immediate cause of the cerebral anæmia in all these cases is doubtful. Mental excitement is usually supposed to lead, in fainting, to a contraction of the minute cerebral arteries. A sudden diminution of the vascular tone of the splanchnic nerve, however, probably occurs in many of the cases. A large part of the blood pours into the dilated vessels of the abdominal viscera, and marked anæmia of the brain results. In some cases primary cardiac weakness may also play a part.

The symptoms of an ordinary fainting fit are known to every one. There are usually certain prodromata. The person begins to "feel badly." Dizziness comes on, the senses are confused, the patient gapes, the ears ring, there are spots before the eyes or total darkness, the floor seems to move, and surrounding objects begin to spin around. All this is almost always accompanied by nausea, and sometimes there is actual vomiting. If the person can lie down promptly the attack is sometimes averted without complete loss of consciousness. Otherwise there is unconsciousness for a time varying from some minutes to even a half hour or longer. What is most noticeable, even in the beginning of the attack, is the pallor, which overspreads the face and often becomes extreme, and is the visible expression of the coincident cerebral anæmia. Often the face and body are bathed in cold perspiration. The pulse is usually small and rapid.

There is no real danger in an ordinary attack. The most important therapeutic measure is to lay the patient horizontal as soon as possible, to favor the return of blood to the brain. Mild stimulants should also be employed; the face should be sprinkled with cold water, the temples rubbed with vinegar or cologne water; brandy or wine should be administered. We can overcome a tendency to fainting fits only by strengthening the constitution.

The results of chronic cerebral anæmia are observable when the cerebral condition is part of excessive general anæmia. Almost all cases of chlorosis, pernicious anæmia, and acute anæmia from loss of blood (as in ulcer of the stomach) display most plainly the symptoms of cerebral anæmia. The phenomena are essentially the same as in syncope, only less in degree. Consciousness is maintained, except in the worst cases. A sort of persistent drowsiness, however, often attended by frequent gaping, is one of the most constant symptoms. The patient is most distressed, as a rule, by loud tinnitus aurium, persistent nausea, and sometimes by obstinate headache. All these symptoms are aggravated, and often increase to an actual attack of fainting, if the patient sits up in bed, and are least marked when he lies as quietly as possible in a horizontal position. The treatment of this condition is, of course, identical with that of the causative disease and the general anæmia.

Cerebral hyperæmia, like cerebral anæmia, may be either chronic or paroxysmal. Of the chronic variety we know almost nothing. It seems doubtful, although it cannot be entirely disproved, whether there is really a "general plethora," or whether the headaches and vertigo of which "full-blooded" persons complain are due to hyperæmia of the brain. Nor have we any direct proof that the cerebral symptoms resulting from chronic poisoning (alcohol, tobacco, etc.), or from persistent mental overexertion, are brought about by hyperæmia, as some assume, rather than by functional disorder of the nervous elements themselves.

Those attacks which are called a "rush of blood to the head" are often attributed to the sudden onset of cerebral hyperæmia. There is a more or less sudden appearance of general excitement, with a sensation of warmth in the head and neck, strong pulsation of the carotids, a red face, general hyperæsthesia and irritability, headache, vertigo, tinnitus, spots before the eyes, and nausea. An attack lasts half an hour to an hour. The special cause of such attacks, however, is usually obscure. It is, indeed, very probable that in these attacks vasomotor disturbances play a part (dilatation of the cerebral vessels from vascular paralysis or from irritation of the vasodilator nerves); but it is very questionable whether these vascular changes are really the primary diseased state rather than the result of some other different morbid condition. Possibly we have to do sometimes with epileptiform attacks, or more frequently with one manifestation of general nervousness, neurasthenia or hysteria (*q. v.*), with primary mental disturbances (anxious ideas, etc.).

In treating "congestion" we should keep the patient as quiet as possible, with head and shoulders raised; and, secondly, we should endeavor to draw the blood away from the brain. This purpose will be served by hot foot baths, sinapisms applied to the chest and the calves, and such purgatives as senna or aperient waters. It is also beneficial to apply cold to the head and to the heart. Local depletion over the temporal region is only rarely employed at present.

To prevent, as far as possible, the recurrence of the attacks, we must have regard to the whole mental and physical constitution of the patient. We may mention, as of chief importance, dietetic treatment (no alcohol, light, chiefly vegetarian diet) and a course at some watering place (Marienbad, Kissingen, etc.), or cold-water treatment.

CHAPTER II

GENERAL PRELIMINARY REMARKS UPON THE LOCALIZATION OF CEREBRAL DISEASES

(Topical Diagnosis of Cerebral Lesions)

The physiological relations of the brain are such that the symptoms of cerebral disease are determined to a greater extent by the locality than by the nature of the lesion. If, for example, there arises at any place a break in the continuity of the cerebral motor tract, the result, as we already know, is hemiplegia upon the opposite side of the body. The result is precisely the same, whether the interruption be due to a hemorrhage, an abscess, a new growth, or an embolic softening. If in any way the function of the motor fibers is suspended, then the necessary sequence in every case is a paralysis of definite extent and definite characteristics. Much the same may be said of many other symptoms which appear when there is a lesion of one or more definite regions, but which are never referable to a special abnormal process, regardless of the portion of brain thereby affected.

However self-evident these simple statements may appear, it required a long time for them to gain universal acceptance among physicians. The chief cause of this was the conception entertained by the older physiologists in regard to the functions of the brain. Flourens, in 1842, taught that functionally all parts of the cerebrum were alike, and therefore any one part could act vicariously for any other; and this view had numerous adherents among physicians as well as physiologists. It was nevertheless experience at the bedside and the autopsy table which first led to observations and discoveries irreconcilable with this view. Above all, it was the lesions found in aphasia which forced men to localize one particular cerebral symptom as due to an affection of one particular spot in the brain. In 1861 Broca announced that the appearance of aphasia is due chiefly to a lesion of the left third frontal convolution; and this was the starting-point of the doctrine of localization in general. Nine years later (1870) appeared the famous treatise of Fritsch and Hitzig, detailing successful attempts at excitation of the surface of the brain in animals, and thus overthrowing the old idea that the gray cortical substance could not be irritated. It was shown that excitation of certain places in the cortex is followed by muscular contractions in well-defined portions of the opposite side of the body, so that we are justified in assuming the existence of a number of cortical centers which are quite limited in extent. These results were soon confirmed by numerous observations in cerebral pathology in man; and to-day our information about the motor functions of the cerebral cortex forms the best-known portion of the doctrine of cerebral localization. Of late years successful work has been done in this exceedingly difficult field by Meynert, Flechsig, and Monakow among anatomists; Ferrier, Munk, Goltz, and other physiologists; and such pathologists as Charcot and his pupils, Nothnagel, Wernicke, Hughlings Jackson, Horsley, and others. It is true that we are only just beginning to know something about the subject. There are numerous contradictory views of cerebral pathology asserted, and numerous questions unanswered. The following summary, therefore, is to be regarded merely as expressing the prevailing opinions now existing. Much in it will surely be altered in the course of time; but still this doctrine of special localization of the various cerebral functions marks out in general outlines the only foundation upon which we can hope to erect a system of cerebral pathology and diagnosis. In the following sketch we shall, for practical reasons, put the results of clinical observations upon man in the foreground, and merely speak incidentally of the corresponding experimental achievements. This will be the quickest way to gain acquaintance with the practical points in the diagnosis of what Griesinger called the "focal diseases"; and then, when we take up the separate varieties of cerebral disease, we shall be able to refer to these general remarks.

1. THE MOTOR REGION OF THE CORTEX CEREBRI

Clinical observation and the results of experiment both teach that a part of the cerebral cortex is distinct from the rest, inasmuch as it is the exclusive seat of motor functions. This "motor region" (*vide* Figs. 201 and 202) is made up chiefly of the anterior central convolution (*gyrus centralis anterior,* in Fig. 201), and the paracentral lobule (*vide* Fig. 203), which lies on the median surface of the cerebrum. To a slight degree the motor region also embraces the neighboring portions of the frontal convolutions. The posterior

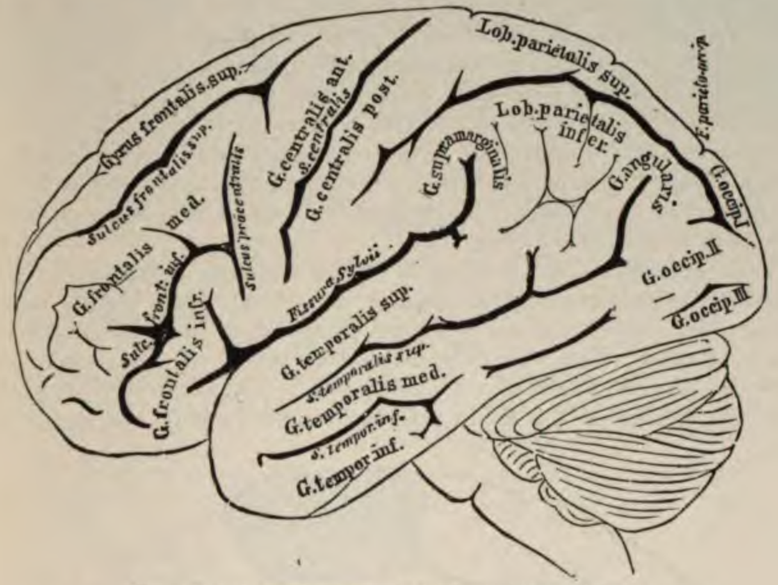

FIG. 201.—Lateral aspect of the brain (from ECKER).
The gyri and lobules are in Roman type, the sulci and fissures in italics.

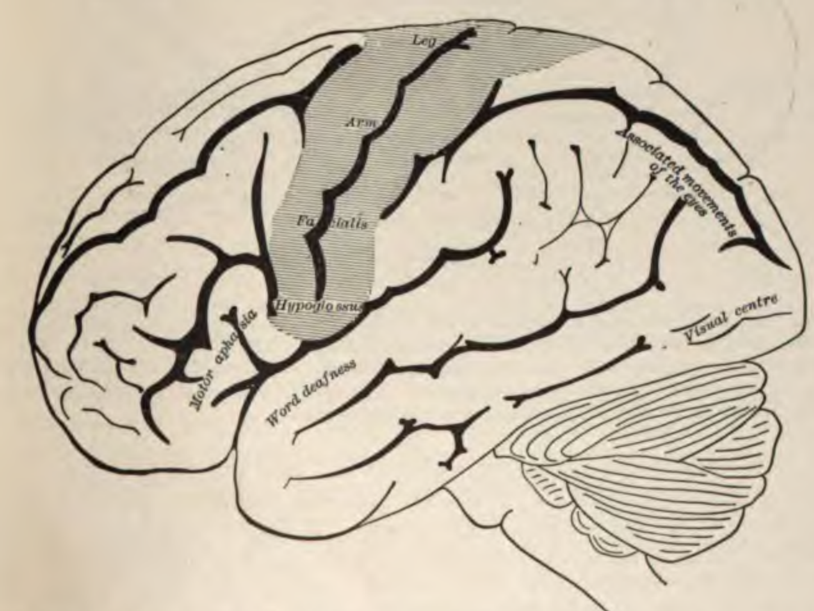

FIG. 202.—Lateral aspect of the brain (after ECKER). The motor region of the cortex, consisting of the anterior central convolution, as well as of the paracentral lobule shown in Fig. 203, and possibly also the posterior central gyrus, is shaded.

central convolution also probably belongs in part to the motor zone; its importance, however, is undoubtedly very much less than that of the anterior central convolution. The entire motor cortical region is also anatomically different from the other regions of the cortex, as Betz was the first to point out, for it alone possesses certain large pyramidal ganglion cells. These cells must certainly be regarded as the same motor-ganglion cells from which the fibers of the pyramidal tract arise (*vide supra,* page 194). However extensive the destructive processes which attack other parts of the surface of the brain, provided they do not involve these particular motor convolutions, they cause no paralytic symptoms; while all diseases which destroy any considerable portion of the "motor" region inevitably result in a motor disturbance on the opposite side of the body.

We can distinguish still further in different regions of the cortex special centers for the various groups of muscles, but we must take special note that the individual centers are not sharply distinguished from one another, but that they overlap. Thus the different centers which are associated have an intimate connection, which also seems necessary physiologically. The center for the movements of the facial muscles (facial region) lies, as it would seem, at the lower end of the central convolutions, and particularly of the anterior central convolution. Near by, probably somewhat lower, is found the center for the movements of the tongue. This center probably extends to the foot of the third frontal convolution, so that on the left side it is accordingly in close proximity to the special motor speech center (*vide infra*). The center for the movements of the arm lies somewhat higher than the center for the facial, occupying roughly the middle portion of the anterior and probably the posterior central convolutions. The center for the lower extremity is

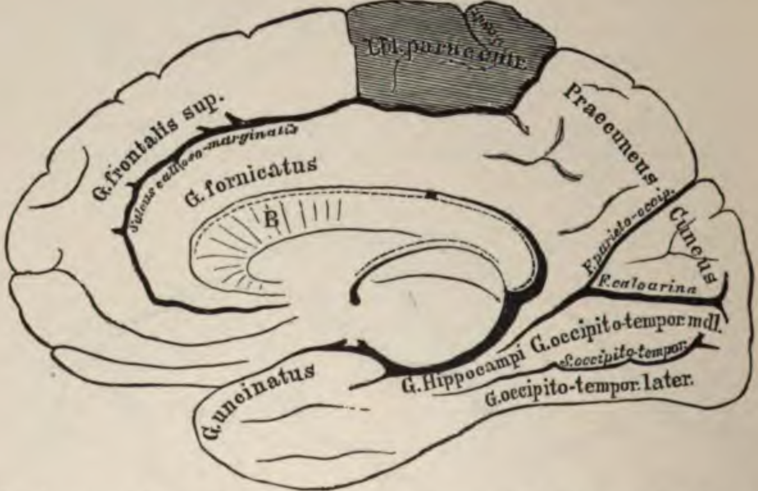

FIG. 203.—Aspect of the median surface of the cerebrum, which is shown when the two hemispheres are separated from each other by a sagittal section. B. Corpus callosum. The differences in the type have the same meaning as in Fig. 201. The paracentral lobule, as a part of the motor region of the cortex, is shaded. (Copied from ECKER, only the paracentral lobule is made more sharply prominent than in the original.)

found partly in the uppermost portions of the central convolutions, but apparently lies for the most part in the paracentral lobule. The center for the movements of the trunk probably lies in front of the leg center in the most posterior portion of the upper frontal convolution and also on the median surface of that convolution (the so-called marginal gyrus, the median portion of the upper frontal convolution).

Within these great cortical centers for the chief portions of the body (leg, arm, face, tongue) there is a much more accurate localization for individual groups of muscles, and perhaps even for individual muscles. This fact has been established especially by the interesting experiments of excitation of the cortex of monkeys by Ferrier, Horsley, Sherrington, and others. Pathological observations and occasional attempts at excitation of the cortex, which have been practiced on man after trephining or in extensive cranial defects arising from injury or disease, have shown that the conditions in man are in general quite the same as in the higher apes. Within the leg center lie special centers for the toes and foot, chiefly above and, according to some authors, especially in the posterior central convolution. Then come the centers for the movements of the foot, the knee, and then those for the movements of the hip. In the paracentral lobule are found chiefly the centers for the thigh and the pelvic and glutæal muscles. Within the arm center and for the most part above—that is, toward the leg center—are the centers for movements of the shoulder and elbow. Farther downward come the centers for the movements of the hand and fingers, chiefly in the anterior central convolution. The separate position of the center for the thumb is especially noticeable; this is in the

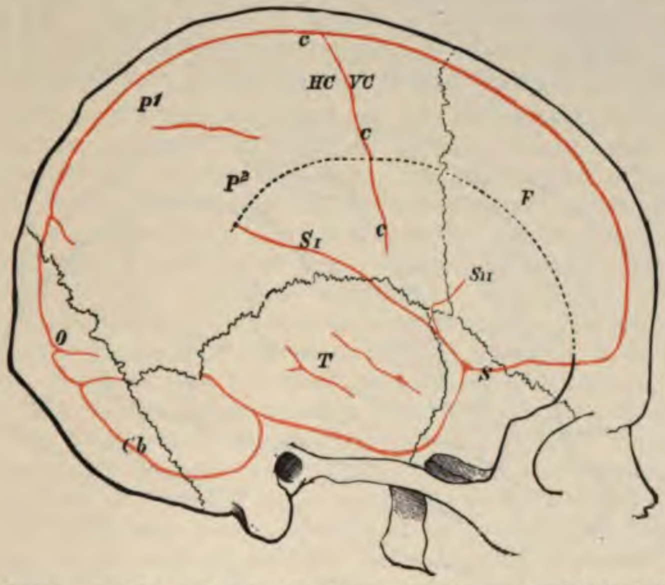

FIG. 204.—(Drawn according to ECKER.) Explanation of the topographical relations between the surface of the brain and the skull. *c*. Fissure of Rolando. *HC*. and *VC*. Posterior and anterior central convolutions. *S*. S_{I} and S_{II}. Fissure of Sylvius. P^1 P^2. Upper and lower parietal lobes. *O*. Occipital lobe. *Cb*. Cerebellum. *T*. Temporal lobe. *F*. Frontal lobe.

posterior central convolution, adjoining the centers for the facial muscles. Farther downward lie the centers for the upper facial territory above the centers for the muscles of the mouth and lips. In the lowest part of the central convolutions and in the adjacent portions of the two lower frontal convolutions lie the motor centers for movements of mastication (the centers for closing the mouth are said to lie above those for opening the mouth), and for the movements of the tongue and vocal cords. The exact position of these centers in man is not yet definitely known. The centers, or, more properly speaking, the connecting stations, for the associated movements of the eyes to one side (fixation movements) seem to be quite scattered. We elicit these movements (usually coincident with a corresponding turning of the head) on irritation of the posterior portion of the frontal lobe (base of the second frontal convolution) adjoining the region of the centers for the arm and face. The principal centers for voluntary movements of ocular fixation (and of turning the head) seem to be located here at the posterior end of the first and second frontal convolutions; but, on the other hand, pathological and experimental observations favor the existence of a center for associated movements of the eyes in the region of the angular gyrus, in the occipital lobe, and other places. This multiple localization is perhaps explained by the fact that the movements of the head and eyes are closely associated both with all complicated activities of the hand and with the functions of sense (listening, looking at objects). The accompanying diagram (Fig. 205), from a drawing by C. K. Mills, depicts the most important localizations in the cerebral cortex which are known at present.

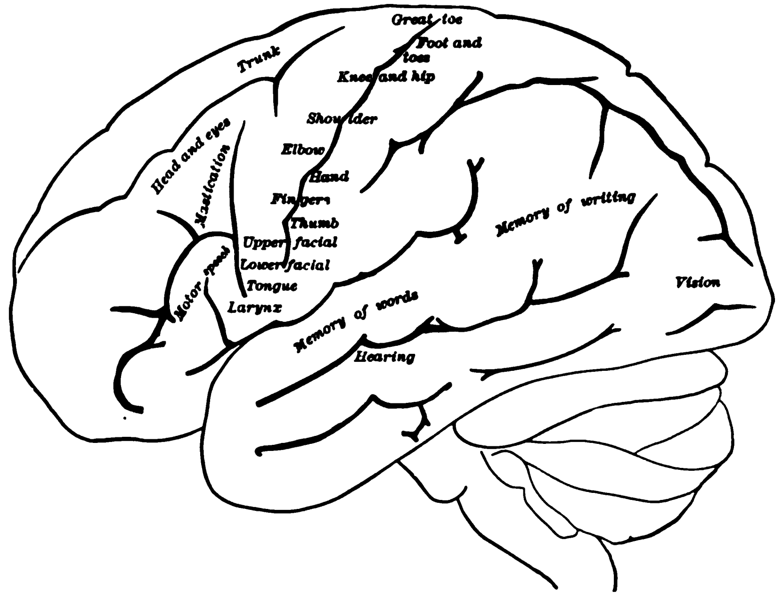

FIG. 205.—Localization in the cerebral cortex.

The topical diagnosis of diseases in the motor region of the cerebral cortex is based upon the following considerations:

In the first place, we have already remarked (page 196) that the relative positions of the motor centers for the face, arm, and leg are such as to allow readily of isolated paralysis of any one of these portions of the body—that is, "monoplegia." In fact, we already possess a long series of observations where circumscribed lesions in the motor area of the cortex produced paralysis of one side of the face, or of one arm or leg, and of no other part. Such paralysis is termed monoplegia of the face, the arm, or the lower extremity. It follows, from what has been said, that even during life we can state, with considerable accuracy, the spot on the surface of the brain where the disease must be situated. Still more frequently, a combined paralysis of two portions of the body is to be observed as a result of cortical lesion; the commonest is a simultaneous paralysis of the arm and face; more rarely we see the arm and leg paralyzed together. On the other hand, we may feel certain, from the position of the motor centers, that no single center of disease could paralyze simultaneously the leg and the face, while the arm escaped injury. As a matter of fact, no such combination has ever been observed. It is also worthy of note that the muscles that always act bilaterally (frontal muscles, muscles of mastication, eye muscles, pharyngeal muscles, vocal-cord muscles, and trunk muscles) have also a bilateral innervation. From each corresponding center, fibers pass not only to the muscles of the opposite side (as is the case in the other centers), but also to the same side. A unilateral focus of disease in any center, therefore, does not cause paralysis of these muscles, while an irritation of these centers always produces bilateral muscular contractions.

Besides this limitation of the paralysis to one portion of the body just discussed, localized disease of the cortex has another very characteristic feature. In it the symptoms of irritation of the motor centers are noticeably frequent. There are tonic and clonic spasms, which, like the paralysis, not infrequently affect one arm, or the arm and half the face. Sometimes, however, they involve the entire half of the body. These paroxysmal spasms are termed "cortical epilepsy," partial epilepsy, or Jacksonian epilepsy; for the movements are just the same as in genuine epilepsy. Numerous cases of disease have taught us that such circumscribed epileptiform attacks occur only in affections of the motor cortex. They furnish still more definite information as to the precise locality of the lesion—for spasms in the distribution of the facial nerve imply that the lower third of the central convolutions is chiefly affected; of the arm, the middle third; and of the leg, the upper portions of the same. If we have the opportunity to observe such attacks of partial cortical epilepsy accurately, we can almost always determine that the twitching begins in quite a limited muscular territory—for example, in the face, the thumb, etc. The attack often begins with a slight sensory aura (paræsthesia in the part of the body affected); then follows a tonic muscular contraction which soon passes into a decided clonus. From this point the twitchings extend, and always in the order corresponding to the local arrangement of the different centers which lie next to one another. It is therefore always of importance in diagnosis to determine in what group of muscles the twitchings begin, because we can thus decide that the corresponding cortical center is the chief seat of the morbid affection. In the muscles innervated bilaterally (the frontal muscles, the eye muscles, the muscles of mastication, etc.) the convulsions also occur bilaterally. In very severe attacks the spasm finally involves the other half of the body, the irritation being conveyed through the fibers of the corpus callosum to the corresponding cortical motor centers of the other hemisphere. The twitchings usually persist the longest in the muscles first affected. In the attacks of circumscribed cortical epilepsy there is no disturbance of consciousness, but if the convulsion extends to both halves of the body, there is usually loss of consciousness, or at least a decided clouding of consciousness. The individual attacks last only a short time—about one or two minutes. After they cease there usually remains a very transitory motor weakness or even complete paralysis in the muscles most affected, which is in a degree an indication of exhaustion of the affected center. Immediately after the cessation of the convulsions, we have repeatedly been able to demonstrate the Babinski reflex in one leg, and to note its disappearance later on. [Ankle clonus has also been observed after such convulsive attacks.—K.] Temporary disturbances of speech are often associated with right-sided Jacksonian epilepsy. The relation of convulsions to permanent paralysis varies greatly. Often, for example, when there is hemorrhage into the central convolutions, violent unilateral convulsions come on simultaneously with the paralysis. In the case of tumors and other lesions which develop slowly, partial epileptiform spasms will often appear quite a long time before there are persistent symptoms of paralysis. Finally, it is not rare for epileptiform attacks to occur repeatedly in regions that are already paralytic. Either of the occurrences described in the two preceding sentences are particularly strong evidence that the cortex cerebri is diseased. Besides the pronounced epileptic attacks, disease of the motor region of the cortex may give rise to less violent symptoms of motor irritation, such as occasional twitching, rhythmical twitching, tonic contraction, etc.

Concerning the condition of sensation in cortical palsies, much contradiction still exists. The experimental researches of Munk and the anatomical investigations of Flechsig and others have led to the conclusion that the termination of the sensory tract of the lemniscus, the so-called sensory sphere, is in part in the same cortical region as the motor cortical centers. We might, therefore, be inclined to suppose that a disturbance of sensation would invariably accompany cortical paralysis in man also, but about this point clinical observations do not yet give perfectly harmonious results. From our own experience, however, we believe that we can say that sensory disturbances are far more common than was formerly supposed. It is a remarkable fact, however, that the sensibility for pain and temperature is hardly ever affected, but the disturbances involve the simple sensations of touch and especially the muscular sense (sensation of the position and movements of the limbs) and the deep sensations of pressure. This explains the fact that the recognition of objects merely by touch, the so-called stereognostic sense, is frequently disturbed in cortical lesions. When sensory disturbances of this kind exist without marked paralysis of the corresponding limb at the same time, a marked ataxia (cortical ataxia) can frequently be demonstrated in the latter.

2. THE OTHER PARTS OF THE CORTEX CEREBRI, EXCEPT THE CENTER FOR SPEECH

1. **Frontal Convolutions.**—Unilateral disease of the anterior portion of the brain may be quite extensive without causing notable disturbance of any kind.

Certainly the greatest part of the upper two frontal convolutions have no motor functions. It is, however, maintained that the posterior portion contiguous to the anterior central convolution, called the foot of the frontal convolutions, does contain motor centers. We have stated above (page 473) that the motor centers for the muscles of the trunk are situated in the upper frontal convolution, especially on its median aspect. This is the reason why a decided swaying of the trunk in walking and in standing (just as in cerebellar ataxia, *vide infra*) has been repeatedly observed in diseases of the frontal lobe, especially in tumors. The third (lowest) frontal convolution on the left side has, as we shall soon see, a connection with the motor processes of speech.

There is a quite generally accepted opinion that the cortex of the frontal portion of the brain is to be regarded as the "seat of the higher psychical functions." Some few cases are on record where extensive bilateral lesions of these parts had for their only manifestations mental symptoms (loss of mental functions, changes in character, the so-called morbid wittiness, *Witzelsucht*, etc.) without any other disturbances. In general paralysis also, and in other forms of dementia, the predominance of atrophy in the frontal lobe is very striking, so that it is actually very probable that the frontal lobe has a close relation to the higher mental processes. Nevertheless, we cannot emphasize too much the fact that, at present, we have no certain knowledge about the precise form of this relation or of the nature of the physical functions.

2. **Parietal Convolutions.**—We know but little about the functions of the cortex of the parietal lobe, and the symptoms which might indicate disease of that portion of the cerebrum. According to Flechsig, a large part of the sensory tract of the tegmentum ends in the parietal cortex. We might therefore expect decided disturbances of sensibility in diseases of the parietal lobe, but unequivocal clinical and anatomical evidence in support of this hypothesis is still almost wholly lacking. Observations on the occurrence of motor disturbances in diseases of the parietal cortex are somewhat more numerous. Centers for associated movements of the eyes seem to be situated in this region (in the angular gyrus). In diseases of this region we may often observe the symptom of the so-called "conjugate deviation"—that is, the simultaneous turning of the head and eyes to one side. The angular gyrus is also said to contain a center for the ocular portion of the facial (orbicularis oculi muscle) and the levator palpebræ superioris; but of course these statements have not been universally accepted. In lesions of the left angular gyrus, the clinical picture of so-called alexia (*vide infra*) has been observed.

3. **Occipital Convolutions.**—The clinical and experimental investigations (Munk, Hitzig, Monakow, Henschen, and others) have unanimously shown that the occipital portion of the cerebrum contains the cortical center for visual sensations. It is here that the fibers of the optic nerve terminate in the cortex cerebri. A glance at the accompanying diagram (Fig. 206) will make it easy to understand the disturbances of vision which result from lesions of the occipital lobe. *L* represents the left eye and *R* the right, *Ch* the optic chiasma, where, as is now certain, some of the fibers of the optic nerves cross to the opposite side. The fibers (distinguished by a broken line) from the outer or temporal half of each retina extend, without crossing, into the corresponding optic tract (*Tract. opt.*), while those from the inner or nasal half of the retina cross over in the chiasma. The fibers of each optic tract pass

by way of the pulvinar of the optic thalamus, the external corpus geniculatum, and the anterior corpus quadrigeminum to the cortex of the occipital lobe. The right occipital lobe, for example, comes in this way to receive the fibers from the outer half of the right retina and from the inner half of the left. If the right occipital lobe becomes disorganized, then the images formed upon the parts of the retinæ just named, belonging to the left half of the field of vision, are unperceived. With each eye the patient sees only such objects as lie in the right half of his field of vision, and is blind to all that lies upon his left. This sort of visual disturbance, where each eye becomes blind to the same "homonymous" portions of the field of vision, is termed hemianopsia (so-called homonymous lateral hemianopsia). A lesion of the right occipital lobe causes, therefore, left-sided hemianopsia, and *vice versa*, destruction of the left occipital lobe entails right-sided hemianopsia. [In a few cases a loss of vision has been observed in symmetrical quadrants of the visual field in the two eyes (quadrant hemianopsia). Investigations have shown that the special visual center is situated above and below the calcarine fissure on the median aspect of the occipital lobe. The fibers from the lower part of the retina go to the region below the calcarine fissure, those from the upper part to the region above. A lesion limited to the region above the fissure in the right occipital lobe will cause blindness in the upper right-hand quadrants of each retina—that is, the lower left-hand quadrants of each visual field.—K.]

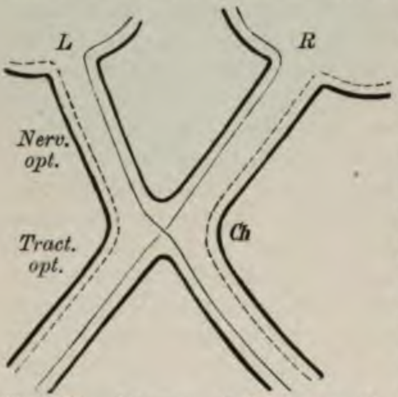

FIG. 206.—Diagram of the course of the optic fibers in the chiasma.

The anatomical evidence is still contradictory as to the precise localization of the visual center. Disturbances of vision are found most constantly in diseases of the cuneus, particularly of the cortex about the calcarine fissure and the first occipital convolution (see Figs. 201 and 203, pages 471 and 472). In this region the sensations of light as such seem therefore to take their rise. Occasionally the color sense is at the same time lost, while in other cases it is preserved. In more extensive lesions, and particularly in bilateral lesions of the occipital lobes, very peculiar visual disturbances occur, which, however, all require further careful study (loss of optical orientation, loss of visual memory pictures, loss of perception of depth, etc.). The symptom of so-called "soul blindness" is particularly interesting (Munk). This condition is when the patient can still see objects, but cannot recognize what they signify. He no longer knows what to do with them, or he uses them in the most utterly wrong way. He no longer recognizes his acquaintances by their appearance, but only by the sound of the voice. In diseases of the left occipital lobe the connections between the optic impressions and ideas of language may sometimes be solely affected; then there develop optic aphasia and alexia; the patient recognizes objects, but he cannot name them. He cannot read or copy, but he can write very well spontaneously and from dictation (compare the following section).

4. **Temporal Convolutions.**—The relation of the temporal lobe to hearing is apparently analogous with that of the occipital lobe to vision. Destruction of the posterior half of the superior temporal convolution, according to some observations, is apparently capable of causing actual deafness in the opposite ear; but the trouble usually disappears in a very short time. Each auditory nerve is therefore probably connected with both temporal lobes. It is much more certain than these observations of actual disturbance of hearing that diseases of the [left] superior (first) temporal convolution cause so-called word deafness ("soul deafness")—that is, the loss of comprehension of spoken language. We will return again to this condition, which is frequently associated with motor aphasia.

We may observe, in conclusion, that the olfactory nerve probably ends in the gyrus uncinatus and the parts adjacent (Fig. 203, page 472), so that we may assume that that is the center for smell. The center for taste is said to be located at the anterior end of the gyrus fornicatus; but, according to other statements, the gyrus fornicatus is said to be the center for pain sensations.

[The sensori-motor centers in the Rolandic region, and the special centers for vision, hearing, and smell which have just been described, occupy only about one third of the area of the cerebral cortex. Flechsig has found that the fibers connected with these areas assume their myelin sheaths at an early period of fetal life, while the fibers connected with other regions become myelinized much later. These other regions he regards as association centers, and believes that they are especially concerned in the higher intellectual functions. The anterior association centers, embracing the anterior portions of the frontal convolutions and the gyrus rectus, are supposed by him to be the chief centers for ideas of personality. The posterior association centers include the præcuneus, the parietal convolutions (except the posterior central convolution), all portions of the occipital cortex except the visual area, and all portions of the temporal cortex except the auditory and olfactory areas. These centers are supposed to be the centers for ideas referring to the external world. The island of Reil and the parts adjacent form the middle association center. Flechsig's hypotheses have been strongly opposed, especially on the anatomical side. It is doubtful whether the projection and the association centers in the cortex can be so definitely distinguished, but the hypotheses thus advanced have also some clinical and pathological support, and they present a rich field for further investigation.—K.]

3. THE CENTERS OF SPEECH AND THE DISTURBANCES OF SPEECH

(Aphasia and Allied Conditions)

The Various Forms of Aphasia and their Anatomical Localization.—For the better understanding of this extremely interesting subject, it is necessary that we should enter somewhat minutely into the processes connected with normal speech.

Incitement to speech—that is, to the oral expression of our thoughts to others—comes either from an internal impulse or from external causes which excite this impulse. Speech always requires internal mental activities—the presence of ideas and their transformation into that which we wish to communicate by speech. Where there are no conceptions there can be no words. The demented person is silent, because, like the newborn infant or the brute, he has nothing to say; but, on the other hand, the impulse to speak must also be present. In melancholic patients we sometimes observe persistent loss of speech, not from any lack of something to say, but because there is no incitement to the act, or because inhibitory influences immediately repress any tendency to utter words. If we take for granted that the mental material for speech exists, then the transformation of this material into actual speech is a result of the following complicated processes, the disturbance of which, individually, produces the various forms of aphasia.

In the first place, the speaker must be acquainted with the word which expresses the mental conception. If, for instance, he wishes to tell another the name of some animal, he must know the appropriate word—"dog," "sparrow," "frog." This knowledge, which, as far as our mother tongue is concerned, we all acquired in childhood, may, as experience shows, be lost again in case of cerebral disease. Just as we may ourselves forget a word momentarily, or as any healthy person may, at the sight of an animal that is perhaps rather rare, be "unable at the minute to think of its name," so in disease one may forget all or a greater or less number of words. Such a patient sees a dog, and knows well enough that it is an animal possessed of such-and-such qualities, but he has forgotten its name. The association between the conception "dog," and likewise between the perception of a dog by the eye and the appropriate vocal representation "dog," is lost. This condition is termed amnesic or sensory aphasia, because it is due to complete or partial loss of the memory for words. The patient knows perfectly well what he wishes to say, but the words escape him. At the same time, in cases of pure amnesic aphasia, the power of repetition is unimpaired. As soon as we say "dog" to the patient, he repeats the word perfectly well, and sometimes he also perceives that this is really the correct word; but in other instances, although the word is correctly repeated, the patient does not become conscious of its meaning (*vide infra* "word deafness").

If we examine cases of amnesic aphasia more carefully we may notice by what special sensory perceptions the memory of the lost word can be eventually called back. Although the mere idea alone does not call the proper word back to consciousness, the sensory perception (so far as we have to do with external objects) may often by association recall the corresponding word. It is, of course, most frequently the visual perception which recalls the memory of the word. If this association also fails we speak of an optic amnesic aphasia. In other cases auditory impressions (e. g., the ticking of a watch, the note of a musical instrument) may restore the recollection of the proper word; or, in other cases still, sensations of touch, taste, or smell. All these associations between given perceptions and the corresponding verbal representations may be destroyed at the same time or one by one, or they may be partially retained. Sometimes it is only by the simultaneous action of different sensory impressions—e. g., perceptions of sight and touch—that the idea of the word can again be recalled to consciousness.

Of great interest are certain cases where there is only partial amnesia. These have been repeatedly observed. Thus a patient forgot nothing but his own name, remembering all other words perfectly; or the loss of words may be confined to but one language, the patient being still able to express himself tolerably well in another tongue. In a case observed by Graves, the patient still knew the initial letter of all words; if, for example, he saw a cow, he would know that the corresponding word began with C, and would look under C in a dictionary till he found the word.

Among the amnesic disturbances, we may also class the dissolution of closely associated sequences of words, as are found, for example, in the order of numbers, in the names of the days of the week, the names of the months, familiar prayers, etc. Although such sequences of words which have been practiced from childhood may on the one hand (*vide infra*) greatly facilitate the pronunciation of the individual words, on the other hand the connection often becomes so loose that the patient may repeat the whole list very well after any person, but he cannot recite it spontaneously. Such tests often show most interesting peculiarities. Thus, we have repeatedly noticed, for example, that aphasic patients can repeat the names of the days of the week very well in their ordinary sequence, but that it is very hard to say them backward. Many patients find great difficulty in counting backward.

If the memory for words be retained, the next requisite for speaking is the transfer of the word image into such action of the muscles of our organs of speech as is calculated to produce the word in question as an actual sound. This motor process is so complicated that an extremely accurate coördination of movements is demanded for the correct pronunciation of the word. Man therefore possesses a separate center, in which this transfer of the word image into the motor processes of speech takes place. If this center be diseased, there again results a loss, or at least a greater or less impairment, of speech. The patient is in this case well aware of the word he wishes to say, but he cannot pronounce it. He has, if we may use the expression, forgotten the movements that are essential to speaking. His tongue and lips are not really paralyzed, but the patient no longer knows how to make use of them for talking. He has reverted to the condition of childhood, before he had learned to talk. The patient often makes the greatest effort to speak. The word he wishes to utter "keeps hovering before him"; he moves his mouth in the most striking manner, but brings out only an occasional sound, and that incorrect. This commonest form of speech disturbance is known as motor (or ataxic) aphasia. Of course it is equally impossible for the patient to repeat a word after some one else. He keeps his eyes fixed on the mouth of the speaker, and endeavors to imitate the motions of his mouth, but he is either partially or totally unable to reproduce the sound.

Motor aphasia exhibits many degrees of intensity. On the one hand there are cases of complete aphasia, where the patient can utter only such separate sounds as "a," "e," etc. And, on the other hand, there are also mild cases where there are merely errors in pronunciation. Such cases may best be termed "ataxic" aphasia. The patient pronounces many words correctly, but with others there are such mistakes as the transposition of individual letters, the misplacement or omission of letters, or, finally, the adding on of letters. For example, he will say "thens" instead of "then," "widow" instead of "window," "dipter" instead of "dipper," "hefd" instead of "held," "wrelstěr" instead of "wrestler," and "belnow" instead of "below." This mildest form of ataxic disturbance is termed "literal ataxia." Patients sometimes affix the same inappropriate termination to many words; for example, a woman used to count: "onen, twoen, threen, fouren, fiven," etc. In most instances the patient can pronounce some words tolerably well, others only imperfectly and with difficulty, and still others not at all. Usually the patient gradually learns a few common words and expressions (e. g., "good morning") by means of persistence in repeating them as they are uttered by another, so that he pronounces them better and better. What is very remarkable, and not so very rare, is that a patient will be able when in a passion to pronounce a word, such as an expression of anger, an oath, or an exclamation, perfectly well, because it is done to a certain extent involuntarily, while he cannot utter the same words if he wishes to say them. One patient under our observation always said "well," in answer to the question, "How are you?" but she was wholly unable to repeat the word "well" by itself when asked to do so. Association also often exerts an appreciable influence; for example, a patient who finds it absolutely impossible to pronounce "six," utters it with perfect distinctness if he begins to count from one, in the ordinary way, up to six. When patients have at last managed to say the right word, they sometimes stick to it—that is, they continue to repeat this word when they should say something else. In many severe cases of motor aphasia the whole capacity for speech is limited to a few syllables or a few brief phrases, which always appear as soon as the patient makes any attempt to speak (so-called *monophasia*). A patient of my own could for a long time utter nothing but the meaningless words, "*selber sag ich nämlich selber*" (self say I namely self). The entire verbal thesaurus of another (female) patient whom I saw consisted of the meaningless sounds "bibi" and "eibibi." Still a third could say only "tinne, tinne." The patient is quite well aware that what he says is wrong, but, despite all his efforts, every attempt to speak excites these same sounds. It produces a comical impression to see the patient use the same invariable word, with all sorts of facial expression. Thus, the woman mentioned above begged for things with "bibi," in a coaxing tone, while sometimes she would give vent to violent anger with a loud "bibibibi."

Amnesic (sensory) aphasia may also vary greatly in degree. It is only rarely observed in its pure form, but it is usually associated with motor aphasia. The amnesic (sensory) aphasia does not always consist of the complete loss of memory of words, but very often merely of a disturbance of the firm bond of association between ideas and their corresponding words. Patients with sensory aphasia therefore very often confuse their words or form entirely false and senseless words (so-called *paraphasia*). Such a patient may talk a long while without conveying any idea to the listener, inasmuch as he says "brush" instead of "bed," or "gove" instead of "give," etc. It is a very interesting fact that in paraphasia the influence of certain associations are often manifest. The patient, for instance, utters a wrong word which has a certain resemblance in sound to the right one, begins with the same syllable, etc. Purely ideal associations sometimes also play a part; thus, one of our patients called a white handkerchief "snow," etc.

The aphasic disturbances thus far described all refer to the power of speech—that is, to the uttering of ideas aloud, but we now come to the description of the disturbance in the understanding of speech, which is often associated with this or which in rare cases may occur by itself. This disturbance consists of the patient's inability to understand the words spoken to him, although he can hear them well. The word when heard no longer excites in his consciousness the appropriate idea. The patient is not really deaf, for he hears everything, but he no longer understands what he hears, and he has forgotten what the words signify. The vernacular sounds to him as a foreign language, of which he knows nothing or of which he has learned but little, would to a well man. This condition is best termed, according to Kussmaul, "word deafness" or "soul deafness." Wernicke also calls word deafness "sensory aphasia," which does not seem to us to be quite correct, since word deafness—that is, the defective understanding of speech—is not identical with the inability to speak in consequence of the loss of ideas of speech (amnesic or sensory aphasia in the stricter sense). Amnesic aphasia and word deafness are, of course, very often associated from allied reasons, but it may very well be the case that a patient has forgotten the word for an idea, but that he at once recognizes its significance as soon as he hears it. Word deafness is in a certain sense a higher degree of verbal amnesia. A healthy person often cannot find the word for an idea in a foreign language, but the idea comes to him at once if he hears the word. It is much harder to speak a foreign language than to understand it.

Slighter degrees of word deafness are very often (we might say almost always) associated with motor aphasia. We must, however, search for them carefully, preferably by giving the patient certain commands (to point to certain objects or parts of the body, to perform certain acts), avoiding any suggestive help by signs or expression, and seeing whether he understands what is said and acts accordingly. We may also test him by holding an object in front of him and calling it by various wrong names and then finally giving it the correct name. We thus see whether the patient gives any sign of recognizing the correct name or not. The detection of word deafness is usually limited, however, to concrete nouns, certain verbs or adjectives, but it is hardly practicable to examine for word deafness for any other words (many abstract words, adverbs, etc.), especially when the patient is also aphasic. We very often notice, therefore, that aphasic patients understand very well the usual brief questions directed to them, but that they are quite unable to follow with complete understanding any long speech, anything that is read to them, etc. They often fail to understand because the rapidity of perception is much impaired. We have seen patients who understood any question or sentence only after the words had been repeated several times. In such patients we also see at times a decided failure of memory of the actual words. If we repeat to them any long sentence, they can repeat only the first four or five words correctly, while they have wholly forgotten the end of the sentence, or at best they have retained the sense but not the sound of the words. We can readily understand how this weakness of memory, or abnormally easy fatigue of memory, may have an inhibitory influence on the understanding of speech.

The list of disturbances occurring in aphasia is by no means exhausted by the examination of the motor power of speech as such and of the understanding of words when heard. Word and idea are yet closely associated with two other modes of expression, which are brought about not by the ear but by the eye. We mean the movements of mimetic expression (or the language of signs in the ordinary sense of the word) and then the far more important written or printed optical signs—letters, script, numbers, etc. Here also we find the close associations between signs and ideas on the one hand and signs and sounds on the other. These associations may be loosened or wholly lost, and then there follow a series of further morbid symptoms which are often to be observed as existing with aphasia.

The disturbances of movements of mimetic expression (*"amimia"* or *"asymbolia"*) have not a very great practical significance, but they often show many interesting features. We are often surprised to see how incapable aphasic patients, who cannot speak, are in giving expression to their thoughts, desires, etc., by signs. They sometimes employ signs in a way that is quite contrary to their usual meaning. Thus, we have often seen patients nod their heads when they clearly wished to deny something. The understanding of signs made by others may also be much diminished.

Agraphia, the disturbance of the power of expression in writing, is far more important than amnesia. We can also distinguish here a pure motor (ataxic) and an amnesic agraphia.

By motor (ataxic) agraphia we understand, of course, not the inability to write on account of any paralysis of the arm, but the inability to unite the different letters correctly to make up the written word. The patient is also unable to copy a word put before him, but he can often do this well when he cannot write spontaneously or from dictation. Then there is writing amnesia (amnesic agraphia). Moreover, we must distinguish between true agraphia (loss of memory pictures for the movements of writing and written words), and secondary agraphia due to word amnesia. If a patient has lost the sound pictures of words, he is naturally also unable to write them. If we test more carefully the ability of aphasics to express themselves in writing, very interesting relations of association can also be shown in this way; the patient may, for example, write a wrong word which in sound or idea is somewhat like the word desired. The entire manner of writing in cases of agraphia is often noticeable. The patient holds his pencil or his pen with peculiar awkwardness; he writes a very small hand, or the letters gradually grow smaller, etc.

Alexia has the same relation to agraphia that word deafness has to aphasia. Alexia is word blindness—that is, the defective understanding of written speech, the inability to read. This disturbance is plainly allied to soul blindness above described (page 478), but alexia may very well exist without special soul blindness, since only the association between the written signs and the corresponding idea has been lost. Patients with pure alexia can neither read nor copy what is written, but they are able to write very well spontaneously or from dictation. Speaking accurately, we must distinguish between reading aloud and reading to one's self. The printed word when seen may excite the idea, although it cannot be pronounced aloud. On the other hand, many patients can read aloud without understanding what is read or pronounced.

In conclusion we may mention briefly an interesting disturbance which is also closely related to aphasia—the disturbance in the power of musical expression [*amusia*]. In these cases the most varied morbid symptoms may occur, which refer both to the perception and to the reproduction of musical tones.

The associations between ideas of tone and the printed notes may be loosened or wholly lost. The examination of the relations existing between words and tones as they exist in singing is also very interesting. We have often seen aphasic patients who could not repeat the words of a familiar song, but who could at once utter the correct words plainly if they sang it.

From all that has been said we can see how extremely manifold and in how many varying combinations the special symptoms of aphasia may occur. Recently aphasia has been subdivided into a large number of varieties, and an attempt has been made to demonstrate a particular localization in the cerebral cortex, or a disease of certain association fiber tracts for each of them. In my opinion, an agreement between the actual conditions, which are frequently very complicated, and all these schematic representations, cannot be realized. We must not conceive that all the separate processes which are necessary in talking and writing, take place in distinct "centers" lying next to each other. We are dealing here with the function of a great mechanism as a whole, all the parts of which are intimately associated with one another, although, naturally, the separate individual tasks are confined principally to a particular anatomical mechanism. The number of possible variations of the clinical picture, however, is so vast that only a careful and detailed study of each individual case of aphasia can give us a complete insight into all existing disturbances.

The Anatomical Changes Occurring in Aphasia.—As has already been mentioned, aphasia was the first cerebral symptom which could be referred to disease of any definite part of the brain. The whole "topical diagnosis" of diseases of the brain has therefore started from aphasia. As early as 1825 Bouillaud affirmed that disease of the anterior lobes of the brain is alone capable of producing disturbances of speech. In 1836 another French physician, Marc Dax, pointed out for the first time that only lesions of the left half of the brain cause aphasia; and in 1861 Broca was able at last to declare that the "center for speech" lies in the left third frontal convolution ("Broca's convolution"). This law has since been confirmed by numerous autopsies,[1] but it applies only to true motor aphasia. If there is motor aphasia, we may with certainty assume that there is disease of the posterior portion of the left third frontal convolution. It is in this region, therefore, that those complicated processes of motor coördination which are essential to the utterance of a word take place. A second important and positive fact we owe to Wernicke: the preservation of the vocal memory pictures of separate words is confined to the left upper temporal convolution. We are justified in accepting this as the "sensory speech center," and in assuming a lesion of this part of the brain in every case of sensory aphasia. Complete destruction of Wernicke's convolution produces word deafness and complete word amnesia.

What disturbances occur if the centers themselves are not destroyed, but the centripetal fibers to the upper temporal convolution, the connecting fibers between this and the third frontal convolution, and the descending fibers from the latter to the lower motor speech centers, is a very interesting but very difficult and complicated question. All speech disturbances of this class have been designated as subcortical aphasias, or, in part also, as conduction aphasias. We are justified in assuming this form of aphasia if an accurate examination of the affected patient shows, not a complete loss of memory for vocal memory pictures, or for the necessary motions for the reproduction of words, but only a failure of reproduction of these memory pictures under certain conditions. Thus, for example, existing word deafness where the ability to understand written language and to write is preserved, can be due only to a subcortical lesion of the conducting mechanism of the temporal lobe, since the ability to read intelligently in itself presupposes the preservation of word-memory pictures. Many varieties of conduction aphasia are produced by lesions in the vicinity of the left island of Reil, for here there are evidently numerous association fibers for the separate speech centers.

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[1] Recently, P. Marie has again disputed the correctness of Broca's assumption. But, up to the present time, his doubts have not been able to destroy the correctness of Broca's doctrine, at least in its main points. [See page 488.—K.]

The understanding of written language may be lost, though the ability to recognize the spoken word and to talk is preserved. In such cases (pure alexia, or so-called word blindness) we may assume a lesion of the lower left parietal lobe. A distinct "writing center" cannot be demonstrated. Writing and reading are functions which do not by any means constitute as important a part of the organization of man as talking. There are countless mentally normal people who can neither read nor write, but not a single one who cannot talk. Not audible speech, but "internal speech," is the necessary result, as well as the necessary premise of incisive intelligent thought. Writing and reading, on the other hand, are acquired useful abilities and activities, which are based on the presence of the speech mechanism. In cerebral affections other organized activities and occupations may be lost just the same as the acquired ability to write. These very interesting disturbances have been designated as apraxia (H. Liepmann).

Finally, we must note that the congenital localization of the separate speech centers in the left cerebral hemisphere applies only to right-handed people. In left-handed individuals the speech centers (though not invariably so) are located in the right hemisphere.

No general rules can be laid down as to the prognosis and course of aphasia, inasmuch as everything must, of course, depend upon the nature of the disease which excites the aphasia. In the aphasias due to cerebral hemorrhage or cerebral embolism (*vide infra*), which are the most common, we very often observe that the disturbances are very marked at first, but that they gradually diminish or wholly disappear. On the other hand, there are also cases of almost complete motor aphasia, which may persist for years until the death of the patient. [When aphasia occurs in childhood it usually disappears completely, perhaps because the other hemisphere of the brain becomes educated.—K.]

In regard to treatment we can here mention only one thing, that methodical training, in the form of actual lessons in speaking or writing, may sometimes be of some service. In motor aphasia we may give lessons, just as to deaf-mutes, by assisting the new practice of the necessary muscular movements by visual impressions, while in verbal amnesia methodical training of the memory is necessary for a new "impression" of the forgotten word. Of course all such training demands much tact and patience on both sides.

[**Dejerine's Views on Aphasia.**—The doctrines of Dejerine and his pupils have acquired so much importance in the study of aphasic disturbances, and these doctrines differ so much from the views expressed above, that it seems advisable to give a brief synopsis of them.

According to Dejerine there are only three centers for images of language: Broca's center, for motor images of articulation, at the foot of the left third frontal convolution; Wernicke's center, for auditory images of words, in the posterior part of the first and second left temporal convolutions; and the center for visual images of words in the angular gyrus. These three centers form the "zone of language," and surround the extremity of the fissure of Sylvius like a horseshoe (see Fig. 205, page 474). They are connected with one another and also with the adjacent sensori-motor center (especially that part which contains centers for the lips, tongue, and larynx), and the visual and auditory centers.

The zone of language may be affected by disease in two ways: (1) The lesion may destroy a part of the zone or its intrinsic fibers; or (2) it may isolate a part of the zone from the adjacent cortex. In the first case—aphasia from lesion of the zone of language, *true* aphasia, or cortical aphasia—internal language as a whole and all the forms of language will be affected to a greater or less extent, although the dominant clinical phenomena—motor aphasia, word deafness, word blindness—will vary with the seat of the lesion. The idea of the word is affected, and this gives rise to a considerable degree of mental impairment.

In *true* motor aphasia from destruction of Broca's center the patient may utter an occasional word (*vide supra,* page 482), but he is unable to do more than that. He has, moreover, lost the idea of the word so completely that he cannot even indicate the number of syllables of which it is composed, and he is wholly unable to write spontaneously or from dictation, except that he can sometimes write his name or some very familiar word. The coexistence of agraphia is characteristic of this form of motor aphasia. There is no marked word deafness, although the spontaneous evocation of auditory images is affected and the patient has difficulty in comprehending rapid speech. Word blindness is less marked, and in copying the patient will copy print as script. In *true* word deafness from destruction of Wernicke's center the patient may seem deaf, confused, or even demented, although the mental failure is much less than at first it appears. There is inability to understand what is said, and the loss of all auditory images deprives the motor center of any regulator, so that the patient uses wrong words, or even mere jargon—paraphasia or jargon-aphasia. There is usually some word blindness and the patient becomes unable to write, or else, on account of the loss of the idea of the word, he may write nonsense or jargon—jargon-agraphia. When he copies he usually makes a servile copy, copying print as print and script as script. In *true* word blindness there is also complete agraphia, and some degree of word deafness and paraphasia. In these sensory forms of *true* aphasia there is less likely to be any paralysis, owing to the situation of the affected centers.

When the zone of language is still intact, but when the lesion has simply cut off the connections of its centers with other centers, we find the condition which Dejerine calls *pure* aphasia, or subcortical aphasia. In these cases internal language is unaffected, the idea of the word is retained, and there is never agraphia. The symptoms are referable solely to the affected center. With *pure* motor aphasia the patient can write normally and can tell the number of syllables in a word. With *pure* word deafness the only symptoms are inability to understand what is said and to write from dictation. With *pure* word blindness there is also right homonymous hemianopsia, and the patient copies in a servile manner, but there is no agraphia; he can write normally, both spontaneously and from dictation. In no form of pure aphasia is there any mental impairment. Dejerine, furthermore, does not believe that there is any special writing center at the foot of the second left frontal convolution or anywhere else; for there is agraphia in every case of lesion of the zone of language, and the agraphia is manifested not only by inability to write with either hand, but by inability to produce words intelligently by arranging lettered blocks or by the use of the typewriter.

Marie's Views on Aphasia.—Marie's views on aphasia require a little further consideration. He has shown that many of the varieties of aphasia which have been described are based upon theoretical considerations, and have no counterpart in clinical experience. He contends that Broca's convolution plays no special part in the function of language, and that the sensory aphasia of Wernicke is always associated with intellectual defect, the disturbance of speech being due more to that than to the involvement of any special speech centers. There is but one form of aphasia, the sensory aphasia of Wernicke due to a lesion involving the left temporal lobe, which is characterized by a disturbance of the general intelligence and by a specialized intellectual deficit of language. If there be in addition a lesion of the lenticular zone (that part of the brain lying inward from the island of Reil), there is also a motor disturbance of speech, anarthria. The left third frontal (Broca's) convolution has nothing to do with the function of speech. The intellectual deficit involves much more than the function of speech if the temporal lobe be affected. There is failure of memory, descriptive gesture, association of ideas, professional knowledge, reading, writing, and the comprehension of written and oral speech. Word blindness and word deafness are simple intellectual troubles in the comprehension of language, and not sensory at all. While these hypotheses are not accepted by the majority of neurologists, it must nevertheless be admitted that the varieties of aphasia that have been described seldom occur in uncomplicated forms, and that intellectual defect is very often present. In the examination of an aphasic patient we should note whether the patient can speak voluntarily, using the right word, whether he can understand what is said, whether he can read intelligently and write; furthermore, we should note whether he can repeat what is said to him, read aloud, write from dictation and copy. It is often the case that patients can understand simple commands, such as a request to put out the tongue or close the eyes, and fail to understand any more complicated sentence. Some patients can respond by shaking or nodding the head, but yet do not respond correctly.—K.]

4. THE CENTRUM OVALE, INTERNAL CAPSULE, CENTRAL GANGLIA, AND REGION OF THE CORPORA QUADRIGEMINA

Centrum Ovale.—The white substance of the hemispheres is made up, so far as we know at present, both of commissural fibers, which connect the various cortical centers together, and of fibers which proceed downward from the centers of the cortex and connect these centers with peripheral parts of the body (corona radiata). As to the symptoms caused by diseases which destroy the commissural fibers, there is hardly anything known. We can only surmise that in case of disturbances of association, such as we have studied under aphasia and kindred disorders, we should consider lesions of commissural fibers, as of those connecting together the temporal and frontal lobes, the occipital and frontal lobes, etc. A break in the continuity of the fibers of the corona radiata must, of course, result in the same symptoms as if the corresponding center were itself destroyed. This explains why circumscribed lesions of the centrum ovale, if they involve the motor fibers of the corona radiata, which proceed from the central convolutions (and only if they do this), cause hemiplegia, or, if very limited, monoplegia. In an analogous manner, disease of the white substance of the occipital lobe may entail hemianopsia; of the temporal lobe, auditory disturbances, such as word deafness. More than once, quite extensive disease of the white substance of the frontal lobe on one side has been discovered postmortem, although no symptoms whatever had been caused by it. Only when the coronal fibers which proceed from the left third frontal convolution are involved in disease is motor or ataxic aphasia inevitable.

Internal Capsule.—The most important facts relating to the functions of the internal capsule, as far as at present known, have been already stated. In particular, it was pointed out that through the posterior limb of the internal capsule, in a comparatively narrow space, passes the pyramidal tract on its way from the central convolutions to the crura cerebri (see Fig. 125, page 193). This is the place where even a very limited focal disease must lead to complete hemiplegia on the opposite side of the body. Clinical experience also shows that the largest number of cases of persistent hemiplegia are occasioned by disease in this spot. The arrangement of the fibers in this region is such that the fibers for the face are, for the most part, anterior; back of them the fibers for the hypoglossus, the fibers for the arm next, and the fibers for the leg posterior. In a brilliant experimental study Horsley and Beevor have shown that in monkeys the different motor portions of the internal capsule can be separately irritated in a much more exact fashion. They found in these experiments that the motor fibers of the internal capsule showed the following arrangement from before backward: Opening the eyes, associated movements of the head and eyes to one side, tongue, angle of the mouth, shoulder, elbow, wrist, fingers, thumb, trunk, hip, ankle, great toe, other toes.

The sensory tract (*cf.* page 156, and Fig. 125, page 193) lies at the posterior extremity of the internal capsule, and apparently includes not only the fibers for cutaneous sensation, but also for the organs of special sense. Complete disorganization of this spot ought therefore to cause, in the opposite half of the body, not only anæsthesia of the skin, but also simultaneously a corresponding impairment of smell, taste, and hearing, and hemianopsia—in short, a so-called complete cerebral hemianæsthesia. Still, in regard to this very point we are much in need of further and definite observations. We have to deal usually only with partial hemianæsthesias, particularly with a combination of hemianopsia and sensory disturbances in the extremities, etc.

Certain practical conclusions in regard to diagnosis can be deduced from the preceding facts. A purely motor hemiplegia, unattended by impairment of sensation, implies a lesion that does not involve the posterior portion of the internal capsule; but probably this portion is also affected, when there is not only paralysis, but considerable sensory disturbance. The sensory disturbance does not invariably extend to all the senses; quite often there is nothing but cutaneous anæsthesia.

Lesions in the neighborhood of the internal capsule may also cause symptoms of irritation of the tracts which pass through the internal capsule. Thus unilateral symptoms of motor irritation in particular have frequently been observed in the form of choreic twitchings (so-called post-hemiplegic chorea, *vide infra*), and unilateral symptoms of sensory irritation (pain, paræsthesia) may also be caused sometimes by lesions in the vicinity of the posterior portion of the internal capsule (Edinger).

Central Ganglia; Caudate Nucleus (Corpus Striatum Proper), Lenticular Nucleus, and Optic Thalamus.—Our knowledge of the physiological importance of the large central ganglia is still very slight. If we find a lesion in the corpus striatum at autopsy in a patient who, during life, showed hemiplegic disturbances, we are always justified in attributing the hemiplegia to an involvement of the closely adjacent internal capsule (*vide supra*).

Scarcely anything is known definitely about the focal symptoms which would indicate a lesion of the lenticular or the caudate nucleus. According to recent experiments on animals, the lenticular nucleus contains centers for regulating heat. The striking changes in temperature sometimes seen in cerebral affections, or the elevations of temperature occasionally observed after apoplexy, could therefore be brought into possible relation to diseases of the lenticular nucleus, but accurate observations are still almost wholly lacking. Vesical control may possibly also depend on the corpus striatum. At least, incontinence of urine has been observed in cases of bilateral softening of this region. But this condition may be interpreted in so many other ways that it does not permit of any definite physiological deductions. [Dana has found softening in both lenticular nuclei in some cases of gas poisoning (carbonic-oxid gas), associated with coma, vasomotor disturbances, and gangrene of the skin—the "syndrome of the corpus striatum."—K.] It is asserted that the optic thalamus is of importance for the movements of mimetic expression (Bechterew, Nothnagel). In hemiplegics we sometimes see that the lower half of the face on one side cannot be moved voluntarily, but it is moved very actively in emotional expression, such as laughing or crying. In such cases we can decide that the thalamus is intact, while in lesion of the thalamus the contrary is observed—the affected half of the face can be moved voluntarily, but it remains completely rigid in emotional expression. As to sensory functions, there is only one which the optic thalamus is certainly known to possess —the central termination of some fibers of the optic nerve lies in its posterior portion (the so-called pulvinar), while other fibers go to the corpus geniculatum externum. Destruction of the posterior part of the thalamus accordingly produces complete hemianopsia (*vide* page 478) of the opposite side. The proximity of the thalamus and the other optic tracts to the posterior part of the internal capsule, in which the sensory tracts run, explains how the symptom-complex of hemiataxia and disturbance of the muscular sense combined with hemianopsia is produced. We have repeatedly observed this clinical picture. The thalamus is also very probably connected with other centripetal fibers from the lemniscus, but we do not know positively whether these are sensory or coördinating fibers, and pathology has not yet taught us as to the functions of the thalamus in this regard. Focal disease of the thalamus has repeatedly occasioned post-hemiplegic symptoms of irritation ("post-hemiplegic chorea," *vide supra*), as is easily to be explained from the propinquity of the pyramidal tract. [Roussy and Dejerine consider that a lesion of the optic thalamus may give rise to a fairly definite group of symptoms—namely: A persistent hemianæsthesia, more or less marked for the superficial varieties of sensibility (touch, pain, temperature), but very pronounced for deep (muscular) sensibility; a slight hemiplegia without contracture, which rapidly disappears; slight hemiataxia and more or less complete astereognosis; severe, persistent paroxysmal pains on the affected side, which are often intolerable, and do not yield to analgesics; choreic or athetoid movements of the limbs on the paralyzed side. The sensory troubles, including ataxia, are due to the thalamic lesion, the motor troubles to an involvement of the internal capsule.—K.]

Corpora Quadrigemina and Crura Cerebri.—Diseases of the corpora quadrigemina are infrequent, and usually, when they do occur, they are merely a part of more extensive lesions of the brain. The anterior corpora are certainly connected with the fibers of the optic nerve. If both of the anterior corpora be destroyed, total blindness is inevitable, while, if only one be disorganized, hemianopsia is to be anticipated. Still, these symptoms are, of course, too ambiguous ever to be regarded as pathognomonic of localized disease of the anterior corpora quadrigemina. Another point to be considered, particularly whenever the corpora quadrigemina are diseased, is the position of the nuclei of the oculomotor and trochlear nerves. This explains why unilateral, or even bilateral, paralysis of the oculomotorius has been repeatedly observed in connection with lesions of the corpora quadrigemina, as have also more rarely nystagmus and immobility of the pupil.

It is to be noted that the nuclei of the oculomotor nerve (*vide* Fig. 197) have a fairly extensive distribution, and that undoubtedly the different ocular muscles have individual nuclei. Particularly important is the differentiation of the most anteriorly situated small-celled nuclei for the internal ocular muscles (muscle of accommodation and the sphincter of the iris) from the large-celled oculomotor nucleus for the external ocular muscles, which is located farther back. This is the reason why, in oculomotor paralysis of nuclear origin, palsies of individual muscles, and particularly isolated palsies of the external or internal ocular muscles, may occur, which is scarcely possible in lesions of the oculomotor nerve. The lateral of the two small-celled anterior nuclei (so-called Westphal-Edinger nucleus) probably innervates the sphincter of the iris and the median small-celled nucleus, the ciliary muscle —i. e., the muscle of accommodation. We must therefore look for the center of the pupillary reflex in the Westphal-Edinger nucleus. Moreover, the motor nerves for the sphincter of the iris and the ciliary muscles do not go directly to these muscles, but are first interrupted by the cells of the ciliary ganglion. In the extensive cellular column of the oculomotor nucleus for the external ocular muscles ("large-celled lateral nucleus"), the center for the levator palpebræ superioris is situated most anteriorly; the centers for the superior rectus, internal rectus, inferior oblique, and inferior rectus then follow in succession. The fibers from the first two nuclei do not decussate, whereas the nerve fibers for the last three muscles mentioned supply the muscles on the same as well as on the opposite side. Evidently, this arrangement serves to secure the most uniform movements of both eyes. Since, however, in the ordinary movements of lateral visual fixation, the internal rectus, which is supplied by the oculomotor nerve, must act together with the external rectus, which is innervated by the abducens, a connection of the oculomotor nucleus (especially the nucleus for the internal rectus) with the abducens nucleus is still necessary. This connection is known anatomically. It is furnished by fibers which run in the so-called posterior longitudinal tract ("dorsal longitudinal tract"). A further important symptom of disease of the corpora quadrigemina is an ataxia of the trunk, similar to cerebellar ataxia (*vide infra*). This ataxia is in all probability due to a disturbance of the superior cerebellar peduncle coming from the corpus dentatum of the cerebellum, which after decussating goes to the red nucleus (nucleus ruber, Fig. 116, page 155), beneath the corpora quadrigemina, and is further connected with the cerebrum. If we have to do, therefore, with an ophthalmoplegia affecting both eyes, but not completely symmetrical, and manifesting itself especially by symptoms of paralysis of the oculomotor and trochlear nerves, and if this ophthalmoplegia be associated with an uncertain, staggering gait, we must consider disease of the corpora quadrigemina. The posterior corpora quadrigemina and the median geniculate bodies are connected with the acoustic nerve, so that in diseases of this region we may expect disturbances of hearing.

Should the crura cerebri become involved in the disease, the resulting symptoms are often very characteristic of the locality affected; there is paralysis of one side of the body (arm, leg, facial nerve), and at the same time a crossed paralysis (that is, one situated upon the opposite side) of the motor oculi. A glance at Fig. 126 (page 194) will explain this phenomenon. Thus, a circumscribed lesion on the right side would destroy the fibers of the third nerve (III) on that side, and therefore produce a right-sided paralysis of the oculomotor nerves, and at the same time, if extensive enough, the lesion would involve the pyramidal fibers of the right crus, and thus occasion left hemiplegia. That disease of the tegmentum would lead to sensory disturbances, or sometimes ataxia may be taken for granted. Diseases of the tegmental region and its vicinity, therefore, cause a clinical picture which is composed of hemiplegia, tactile hemianæsthesia (or hemiataxia), and crossed oculomotor paralysis of the opposite side.

5. THE CEREBELLUM

The cerebellum is principally an organ for regulating (coördinating) the positions and movements of the trunk. A correct and steady fixation of the trunk is not only the fundamental requirement for all movements of the body as a whole (walking, standing, running, jumping), but also for all the finer movements of the extremities, since any unsteadiness of the trunk would naturally disturb every fine movement of the hands, etc.

In addition, the cerebellum contains exceedingly important mechanisms for regulating the balance of the head and its relations to the positions and movements of the eyes. As we are almost continually changing the position of the head, a separate regulating mechanism is required for these movements. With every movement of the head the relation of the external world to our retinæ is also altered. We would therefore be continually confused in regard to motion and the distance and position of external objects if the movements of the trunk, head, and eyes were not minutely regulated in reference to each other, so that, under normal conditions, the position of our body in space with reference to external objects is always clearly recognized by us.

To accomplish this task the cerebellum must receive, in the first place, reliable information of every condition (position, attitude, movement) of the trunk, and, in part, also of the lower extremities. These reach the former through the centripetal lateral cerebellar tract and Gowers's tract, which, on the one hand, communicate with the posterior spinal roots, and, on the other, are connected with the cerebellum by means of the corpora restiformia. The regulation of the movements of the head and of the consequent movements of the eyes is accomplished by the vestibular mechanism, which starts from the semicircular canals of the labyrinth, and thence communicates the appropriate stimuli by means of the vestibular nerve, to the vestibular nucleus, Deiters's nucleus, and, farther on, to the cerebellum. Deiters's nucleus is, however, intimately associated with the muscles of the eye through the posterior longitudinal tract.

In order to utilize all these centripetal cerebellar stimuli for regulating the movements of the head, trunk, and eyes, centrifugal connecting paths from the cerebellum to the muscles are necessary. These, as far as we know at present, are furnished principally by the peduncles, which extend to the red nuclei, and by Monakow's tract (*vide* page 195), which descends from the latter; furthermore, by the vestibulo-spinal tracts, which descend from Deiters's nucleus, and, lastly, the previously mentioned connection between the vestibular apparatus and the ocular muscles.

Corresponding to these two functions of the cerebellum, which are, of course, closely associated, two characteristic symptoms are found primarily in affections of the cerebellum—namely, (1) cerebellar ataxia, that is, unsteadiness of the trunk in standing and walking, and (2) cerebellar or vestibular vertigo, that is, disturbance of the normal judgment of the position in space of our body with reference to objects of the external world. As a third cerebellar symptom, cerebellar muscular hypotonia may be added.

Cerebellar ataxia affects the trunk and lower extremities. Both standing and locomotion are chiefly interfered with. When the patient is lying in bed he can move his legs almost as well as ever, and with normal vigor; but as soon as he gets up the characteristic motor disturbances become very evident. Even while standing still, the whole body can usually be plainly seen to sway back and forth. This becomes more marked if the patient brings his heels together. If he stands with his legs widely apart, the trouble is less noticeable. Closing the eyes, as a rule, aggravates the swaying only very little, inasmuch as the conscious cutaneous and muscular sensibility of the lower limbs is transmitted to the cerebral hemispheres and remains normal in uncomplicated cerebellar disease. When the patient tries to walk, he sways and totters, precisely as if he were deeply intoxicated, but usually in a very different way from that seen in tabes dorsalis. Instead of the uniform stamping and pitching gait of the latter, cerebellar ataxia causes a staggering of the whole body, so that in severe cases the patient loses entirely the ability to walk straight, but seems to fall forward, as it were, in a zigzag line, now to the right and now to the left. The most pronounced cerebellar ataxia is found in lesions of the vermis. In unilateral cerebellar disease a preponderance of the ataxia in the corresponding (homolateral) side of the body can occasionally be demonstrated. A diminution of the muscular tonus (hypotonia) is almost invariably associated with the ataxia. This is explained by the fact that the muscular tonus is sustained by reflexes which are caused by centripetal stimuli from the spinal cord to the cerebellum. Sometimes, but by no means invariably, it is noticed that in walking the body sways principally in one particular direction, either forward, backward, or to one side. Such peculiarities, however, do not enable us, with our present knowledge, to determine with certainty just what position in the cerebellum the lesion occupies. The most we can do is to surmise, in such a case, that the middle peduncles of the cerebellum (*vide infra*) are involved. It is worthy of note that the ataxia does not involve the upper extremities. Many a patient who can scarcely walk unaided is still able to perform the most delicate manipulations with his hands. This shows that it is only in maintaining the equilibrium of the trunk through the muscles of the pelvis and the leg (in standing, sitting, walking, etc.) that the function of the cerebellum is important. [Babinski has shown that this ataxia is due, at least in part, to the fact that the muscles of the trunk and legs do not act together; that on walking, for example, the legs may tend to go forward while the trunk lags behind, or *vice versa* (cerebellar asynergy). This condition may be limited to one side of the body (hemiasynergy), which is the same side as the cerebellar lesion. Babinski also thinks that in cerebellar lesions, when the patient is in bed, he can keep his limbs in one position longer than a normal person can, a condition resembling catalepsy. He has found also that, with a cerebellar lesion, the patient is often unable to make antagonistic movements, such as pronation and supination, in quick succession (adiadocokinesis).—K.]

As already stated, this cerebellar ataxia is in most cases attended with pronounced vertigo. There is not, however, a complete correspondence between the locomotor disturbance and the dizziness. Exceptionally one symptom may be present without the other. The vertigo is usually felt only when the patient is erect, standing or moving about, and is very seldom felt when he lies quietly in bed. This is explained by the fact that the diseased cerebellar mechanism becomes functionally active, for the most part, only on moving the head or body. If the centripetal vestibular mechanism itself is diseased (Menière's disease, *vide infra*), incorrect perceptions which cause sensations of vertigo are conducted to the cerebellum even when the patient is at rest. Cerebellar vertigo is very frequently combined with nystagmus. But it is a difficult matter to decide to what extent the nystagmus depends directly on an affection of the vestibular mechanism, or on an indirect action (tumors) on the pons or the posterior longitudinal bundle. Cerebellar vertigo is a true vertigo: the patients have the sensation as if they themselves or the surrounding objects were revolving. As Stewart and Holmes have determined, the apparent motion of the objects generally occurs from the affected toward the well side. The apparent motion of the body, on the other hand, is from the affected toward the normal side in intracerebellar lesions, and from the normal to the affected side in extracerebellar tumors of the occipital fossa. The patients are, of course, by no means always clear in their statements on this point. The sensation of vertigo is constantly present, but attacks of more marked exacerbations occur.

Little definite is known about other symptoms of disease in the cerebellum. Many of the morbid phenomena which are associated with the true cerebellar symptoms are explained by the indirect action of the pathological process. Thus, for example, a true ataxia of the legs may generally be due to an indirect action of the disease on the lemniscus. The patellar reflex has sometimes been found absent in cerebellar disease. This symptom also is probably always dependent upon coexisting disturbances of the posterior spinal roots from the increased pressure of the cerebro-spinal fluid. In some cases hemiparesis (asthenia) of the extremities has been found on the side of the disease (corresponding to the results of Luciani's experiments on animals). Possibly this is a direct cerebellar symptom. According to L. Mann, homologous cerebellar hemiparesis is distinguished from crossed cerebral hemiparesis by the fact that it does not follow the type of pyramidal tract paralysis (*vide infra*), but affects all the muscles equally; furthermore, that it is not associated with hypertonia, but with hypotonia, and shows no increase of the tendon reflexes, as well as no Babinski reflex. We will return later to certain special symptoms, especially those occurring in tumors of the cerebellum.

We must add in conclusion a few words about diseases of the middle cerebellar peduncles (crura cerebelli ad pontem). Those peculiar symptoms, known as forced movements and forced positions, are usually referred to an irritation of these peduncles on the ground of physiological experiments. Forced positions are when the patient always lies upon one particular side in bed, whether he is perfectly conscious or in a state of complete unconsciousness. If he is put in any other posture, he at once involuntarily reassumes his former position. Not infrequently this forced position of the trunk is accompanied by a corresponding forced position of the head and eyeballs, while the extremities are seldom affected. Genuine forced movements are seen far less often. If the patient be able to walk at all, they evince themselves as involuntary circular movements ("circus movements"), etc. According to the statements of Russell, the forced movements in disease of the *right* middle cerebellar peduncle are in the direction "of a corkscrew screwed *into* a cork," and, in disease of the *left* peduncle, the direction "of a corkscrew screwed *out* of a cork." It must be said, however, that the symptoms of forced positions and forced movements are on the whole rare and of doubtful value in diagnosis; for in some cases of cerebral disease the same symptoms have been observed without any discoverable affection of the middle cerebellar peduncle. On the other hand, in two cases of tumor of the middle cerebellar peduncle with autopsy we failed to find any forced movements.

For convenient reference we subjoin a summary of the most important facts bearing upon the localization of cerebral diseases.

1. The most frequent cause of ordinary hemiplegia is a lesion of the pyramidal tract in the posterior limb of the internal capsule. If the hemiplegia be persistent, then this tract is actually destroyed; if temporary, the tract

has been functionally deranged for a time by focal disease in neighboring parts of the brain.

2. Monoplegic cerebral paralysis is usually due to affections of the cortex of the brain—that is, the central convolutions and the paracentral lobule. Monoplegia of the face and tongue is the result of lesions in the lower extremity of the anterior central convolutions. Monoplegia of the arm is referable principally to some lesion of the middle third of the anterior central convolutions. Monoplegia of the lower extremity implies some affection of the upper portion of the anterior central convolutions and the paracentral lobule.

3. Hemiplegia or monoplegia, if associated with epileptiform convulsions affecting either one half or one particular portion of the body, are almost always caused by cortical lesions. These same symptoms of motor irritation without accompanying paralysis are likewise to be ascribed to some irritation of the above-mentioned regions of the cortex.

4. Hemiplegia with crossed paralysis of the oculomotor nerve indicates a lesion of the crus cerebri. Coexisting tactile hemianæsthesia implies that the tegmentum is involved. Isolated paralysis of the external or internal ocular muscles, supplied by the oculomotor nerve, indicates a nuclear origin of the paralysis.

5. Hemiplegia with crossed facial paralysis implies, with great certainty, that the lesion is situated in the pons. A further sign of a pons lesión (abducens nucleus) is paralysis of lateral associated ocular movements.

6. Post-hemiplegic chorea (*vide infra*) seems to occur especially when there is focal disease in the neighborhood of the posterior part of the internal capsule.

7. Hemianæsthesia of the skin, which may be associated with hemianopsia, is due chiefly to lesions of the most posterior portion of the internal capsule.

8. Hemianopsia may be due to a lesion of the occipital lobe (cuneus). A lesion of the posterior extremity of the internal capsule may also cause it, in which case it is usually associated with hemianæsthesia or hemiataxia. Finally, it may be produced by affections of the pulvinar of the optic thalamus, of one lateral geniculate body, of one of the anterior corpora quadrigemina, or of one of the optic tracts.

9. Genuine motor aphasia indicates disease of the foot of the third left frontal convolution.

10. Word deafness (loss of understanding of speech) is due to disease of the first left temporal convolution; word blindness (loss of understanding of writing) is due to disease of the left lower parietal lobe (angular gyrus).

11. Difficulty in articulation implies disease of the medulla, as does also dysphagia.

12. Staggering gait (ataxia of the trunk) and true vertigo are the most constant symptoms of cerebellar disease, but ataxia of the trunk may also occur in diseases of the corpora quadrigemina and of the frontal lobe (*vide supra*). Forced positions and forced movements perhaps indicate lesions of the crura cerebelli ad pontem.

As an appendix, we may add a very brief review of the course of the most important conducting tracts:

1. **Motor Pyramidal Tract.**—(*a*) *First central neuron:* Ganglion cells in the motor cortex of the brain, axis-cylinder processes through the corona

radiata, posterior limb of the internal capsule, crusta of the crus cerebri, decussation in the pyramids, postero-lateral column of the spinal cord, terminal arborization in the anterior horns (for the motor cranial nerves previously in their nuclei). (*b*) *Second peripheral neuron:* Ganglion cells in the anterior horns (or in the nuclei of the motor cranial nerves), axis-cylinder processes in the anterior root and the peripheral nerve, terminal arborization in the muscular fibers.

2. **Chief Centripetal (Sensory and Coördinating) Tract.**—(*a*) *First (peripheral) neuron:* Ganglion cells in the spinal ganglion, axis-cylinder processes partly in the peripheral sensory nerve [modified protoplasmic processes], partly in the posterior root. The posterior root fibers in part split up immediately about the ganglion cells of the posterior horn, and in part continue upward in the posterior columns of the same side to the nuclei of the columns of Goll and Burdach. The physiological significance of these two kinds of fibers has been previously explained (*vide* page 156). (*b*) *Second neuron:* Ganglion cells in the posterior horns or in the nuclei of the columns of Goll and Burdach, axis-cylinder processes through the (crossed) antero-lateral columns, interolivary layer, lemniscus tract of the medulla and pons, tegmentum of the crus cerebri, posterior portion of the internal capsule. Terminal arborization in part in the optic thalamus. (*c*) *Third neuron:* Ganglion cells in the optic thalamus, axis-cylinder processes to the cerebral cortex (central convolutions, parietal lobe).

3. **Chief Optic Tract.**—First peripheral neurons lie wholly in the retina, and the ganglion cells of the second neurons are also in the retina, axis-cylinder processes through the nerve and optic tract (partial decussation!) to the lateral geniculate body, the anterior corpora quadrigemina, and the pulvinar of the optic thalamus, where the terminal arborization takes place. Third neurons from the cells of the parts mentioned through the posterior portion of the internal capsule [and the optic radiations of Gratiolet] to the cortex of the occipital lobe (cuneus).

4. **Chief Acoustic Tract.**—The ganglion cells of the peripheral neuron lie in the cochlear ganglion of the cochlea, the axis-cylinder processes in the cochlear nerve, termination in the ventral acoustic nucleus. Here the second neuron begins, whose axis-cylinder process passes through the trapezoid body and the striæ acusticæ (?), and then through the lateral lemniscus to the posterior corpora quadrigemina and the median geniculate body. Here probably a third neuron begins, which ends mainly in the cortex of the temporal lobe. The acoustic fibers also run partly crossed and partly uncrossed, so that each auditory nerve is connected with both hemispheres.

CHAPTER III

CEREBRAL HEMORRHAGE

Ætiology.—The cause of cerebral hemorrhage should always be sought in some disease of the coats of the minute cerebral arteries. Cerebral hemorrhage occurs most frequently, therefore, in persons who suffer from general arteriosclerosis, and especially from arteriosclerosis of the cerebral arteries;

but other forms of disease of the walls of the blood vessels, fatty and hyaline degeneration, and, in particular, syphilitic endarteritis, may give rise to cerebral hemorrhage. In 1868 it was first shown by Charcot and Bouchard that in many cases of large cerebral hemorrhage there are miliary aneurisms of the small atheromatous arteries of the brain substance, some one of which has burst and allowed the blood to escape. All later investigators have confirmed their statements about the occurrence and importance of these miliary aneurisms. The aneurisms may attain a diameter of a millimeter or more. They usually appear like spindle-shaped dilatations of the entire circumference of the vessel, although sometimes the bulging is confined to one side of it. Inasmuch as the intracerebral arteries possess almost no true adventitia, it is easy to see that these vessels are especially predisposed to aneurismal dilatation (especially in the form of a so-called dissecting aneurism).

Most of the circumstances which come under our consideration as favoring the development of cerebral hemorrhage have also a close relation to arteriosclerosis. Age plays a specially important part. Although exceptionally a younger individual may be attacked, the majority of sufferers are over fifty years old—that is, at the time of life when arteriosclerosis usually becomes most fully developed. Again, cerebral hemorrhage is decidedly more frequent in men than in women, which is also true of atheroma. Alcoholism, syphilis, and gout are also reckoned among the ætiological factors of both disorders, and in both a hereditary predisposition is not very rarely demonstrable. What is called the "apoplectic habit" also deserves brief mention. Although there is no variety of constitution which exempts its possessor from the possibility of cerebral hemorrhage, yet it cannot be denied that often the victims of apoplexy do exhibit a certain "habit." Such persons are not very tall, but are corpulent, broad-chested, with a short, thick neck and round face; and they have not been disinclined to the pleasures of the table and to alcohol. Such persons often suffer at the same time from emphysema, moderate hypertrophy of the heart, and general arteriosclerosis, as the condition of the radial and temporal arteries may disclose even during life.

Granting, therefore, that disease of the arteries, and sometimes more particularly miliary aneurisms resulting from chronic endarteritis of the smaller cerebral arteries, must be regarded as the chief cause of cerebral hemorrhage, then, on the other hand, the question suggests itself whether an abnormal elevation of the blood pressure may not have some part in determining the hemorrhage. If the coats of the arteries are normal the greatest increase of pressure would not be able to cause rupture of the vessels; but if the wall of the artery is diseased, or if aneurisms have already been developed, then there can be no doubt that a persistent or even a temporary elevation of the blood pressure must favor the bursting of the vessels. In this sense a cerebral hemorrhage, occurring in patients with certain forms of cardiac hypertrophy (contracted kidney, idiopathic hypertrophy, etc.), combined with disease of the vessels, may be referred in part to the increased arterial tension; but it is most of all with regard to many exciting causes, which are immediately followed by a cerebral hemorrhage, that increased blood pressure assumes great importance. Here it is temporary. Cerebral hemorrhage may, for example, follow excessive muscular exertion, the ingestion of a large amount of food, indulgence in alcohol, taking a cold bath, violent mental excitement, etc. An apparently slight injury is especially to be considered. In all such cases, however, the change in the arteries is a necessary prerequisite.

It should be mentioned, in conclusion, that cerebral hemorrhage is sometimes merely the expression of a general hemorrhagic diathesis, as we find it in leukæmia, pernicious anæmia, and those affections which are called, in a stricter sense, hemorrhagic diseases, such as scurvy and purpura hemorrhagica. The grave infectious diseases, including septicæmia, typhus or typhoid fever, and smallpox, may occasion hemorrhage into the brain as well as into other organs. The hemorrhages are generally, however, from capillary vessels, and are very rarely extensive.

Pathology.—Arteriosclerosis and miliary aneurisms do not develop in all the cerebral arteries with equal frequency, and accordingly we find certain regions particularly liable to cerebral hemorrhage, being very much oftener affected by it than others. These regions seem also to be the ones where the arterial blood pressure is relatively the highest.[1] Hemorrhage takes place by far the most frequently from the branches of the artery of the fissure of Sylvius. The large central ganglia in the neighborhood of the lateral ventricles, the optic thalamus, caudate and lenticular nuclei, and also the adjacent white matter of the internal capsule and centrum ovale, are therefore usually affected by the hemorrhage. Hemorrhages in other portions of the brain are much less frequent—such as hemorrhages into the convolutions, the pons, the cerebellum, the crura cerebri, or the medulla. If the blood escapes into the neighborhood of a ventricle, it may burst into the latter. Likewise, in rare instances, an effusion of blood in the cortex may make its way out upon the surface of the brain.

An extensive collection of blood in one of the hemispheres may exercise so decided a pressure upon surrounding parts that the results of increased tension upon the affected side are at once recognized when the skull is opened. The dura on that side is more tightly stretched, the falx is crowded over to the opposite side, the convolutions on the convexity seem flattened, and the furrows are shallow. Exceptionally, when there is a very large effusion reaching nearly to the surface, we may even detect fluctuation.

On cutting through the brain substance we find the seat of hemorrhage, and are enabled to determine its position and extent. Its size, of course, varies considerably in different cases; it may be small, or it may occupy a large part of an entire hemisphere. The wall of the effused mass is made up of ragged and torn cerebral tissue, and the mass itself contains *débris* of the nervous elements entangled in the coagulated blood. The blood clots are almost always very dark-colored when fresh. Later on the mass changes to a chocolate-colored or more brownish-yellow pulp, composed of the disintegrated remnants of the nervous substance and the clotted blood. The microscope reveals, particularly in the immediately surrounding tissues, numerous fatty granular cells. These are white blood corpuscles which have absorbed the fat resulting from the decomposition of the myelin substance. There is also always an abundance of hematoidin crystals, due to the disintegration of the red blood globules. At a greater distance from the effusion the tissues present a yellowish tinge, from the imbibition of such blood pigment as has reached them in a state of solution; and there is also usually an œdematous softening of the parts not too far removed from the hemorrhagic focus.

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[1] [Mendel has found that the normal blood pressure in the lenticulo-striate arteries, which are most prone to rupture, is much higher than it is in the cortical and many other small arteries of the brain.—K.]

If the patient survive, the mass is gradually absorbed. It slowly diminishes in size, and the surrounding parts tend to reassume their normal relations. The final result in many cases is a cavity filled with serous fluid and bounded by smooth walls. This "apoplectic cyst" remains stationary. In some instances, however, and particularly if the effusion be rather small, the walls approach each other as more and more of the fluid is absorbed; there is a great hyperplasia of connective tissue, and so, finally, there is nothing left but a so-called apoplectic scar, usually of a yellow color, due to vestiges of the blood pigment. The position and dimensions of the permanent lesion determine, of course, the question of secondary descending degeneration (*vide* page 429), as well as the nature and extent of the persistent clinical symptoms.

Clinical History.—The symptoms of cerebral hemorrhage agree closely with the anatomical lesions just described. The vascular disease responsible for the hemorrhage (arteriosclerosis, miliary aneurisms) in many cases excites no morbid symptoms; but, on the other hand, we often find that persons attacked by a cerebral hemorrhage have suffered much from vertigo, headache, etc., for more or less time before the shock. Such symptoms are undoubtedly due to the disease of the cerebral blood vessels and to the slight disturbances of the circulation thus produced.

As soon as the vessel wall or an aneurism bursts, however, and blood escapes into any part of the brain substance, there is immediately seen a group of grave cerebral symptoms, collectively termed an apoplectic attack, or "shock." As the blood escapes under a pressure nearly equal to the general arterial pressure, and doubtless much greater than that to which the soft substance of the brain is normally exposed, the affected portion of the brain is at once subjected to a considerable increase of tension, which is transmitted for various distances in all directions. It need not be said that the destructive influence of the hemorrhage, which is probably manifested by a compression of the nervous tissue itself and also more especially by a compression of the blood and lymph channels and a consequent disturbance of the circulation, may vary exceedingly; and that therefore the symptoms are by no means equally severe in all cases. The larger the rent in the blood vessel, and the more rapid and abundant the consequent hemorrhage, the worse is the apoplectic attack. Bleeding from larger vessels is therefore usually attended by graver symptoms than from the minute arterial twigs. An extensive cerebral hemorrhage sometimes causes the patient to fall down suddenly in complete unconsciousness, while smaller hemorrhages may occasion only a temporary attack of vertigo and slight cloudiness of intellect. If the tear in the wall of the artery be very small and narrow, permitting the blood to escape but slowly, then there may be no sudden attack at all, the phenomena requiring a certain length of time for their development.

There is also an important relation between the location of the hemorrhage and the severity of the apoplectic attack. The chief symptom of these cases is loss of consciousness (about which we shall soon speak at length); and as this is certainly due to an interruption of the functional activity of the cerebral cortex, it is plain that the nearer the cortex is to the hemorrhagic focus, the more apt are the symptoms to be serious. It is confirmatory of this that hemorrhage into the more deeply situated portions of the brain, the crura cerebri or the pons, quite often occasions comparatively slight symptoms. But there is a fact about the circulation in the brain that often causes the shock from hemorrhage into the brain stem to be greater than the shock following hemorrhage into the cortex or the white substance of the hemispheres. This fact is that the brain stem has comparatively much larger arteries than the other parts just mentioned, which contain only minute blood vessels. Furthermore, as Duret and Heubner have shown, the blood vessels are so distributed that the arterial tension in the brain stem is not a little higher than in the other portions. This renders intelligible the clinical phenomenon that hemorrhages in the territory of the main arteries, besides being, as we have said, the most frequent of any, produce apoplectic symptoms even when the effusion is comparatively small; while sometimes hemorrhages of about the same size in the cortex or white substance may not be noticed.

The clinical phenomena of the apoplectic attack will now be considered in detail. The onset is sometimes absolutely without warning, but in other cases it is preceded for a greater or less length of time by certain prodromata. These are either the result of the disturbance of circulation caused by the disease of the blood vessels in the brain, and then, as already stated, they comprise occasional headache, vertigo, tinnitus aurium, spots before the eyes, languor, and muscular weakness; or they are caused by minute hemorrhages, which seem not infrequently to precede a greater one. In such a case, the friends state that of late the patient has had one or more slight and brief attacks, characterized by faintness, temporary trouble in speaking, sudden but temporary weakness of an arm or leg, and similar symptoms. The prodromata may extend over several days or even weeks and months preceding the severe attack.

In other cases there are no such premonitory symptoms. The apoplexy occurs unexpectedly and suddenly. In the midst of apparently vigorous health the patient sinks down "as if he had been struck." In still other cases there are indeed no prodromata, but the symptoms do not at first appear in all their severity, and occupy some hours or even a whole day in their gradual development. This is due to a slow and gradually increasing hemorrhage, and is termed a slow or delayed apoplectic attack. The patient grows confused, anxious, and delirious (a case of our own had pronounced hallucinations of sight), the arm and leg on one side become paretic, and gradually more and more completely paralyzed, and after a few hours complete unconsciousness comes on.

The attack may be rapidly fatal. In such cases the abnormal pressure probably involves the medulla oblongata and paralyzes the cardiac and respiratory centers there situated. Usually, however, there is merely a complete loss of consciousness, more or less rapidly developed. Sometimes the patient has time to lie down. He usually sinks back in his chair or falls to the floor, and becomes deeply comatose. The face is often noticeably flushed, and the pulse full and tense, but not infrequently somewhat slow, because of the increased cerebral pressure. The respirations are deep, noisy, stertorous, and likewise often slow. The relaxed cheeks and lips are often drawn deeply in at every inspiration, and puffed out at every expiration. The temperature is usually subnormal at first, later regaining the normal, or even a higher point. In a rapidly fatal case, however, the temperature remains depressed till death. It is not very rare in severe cases to observe a peculiar position of the head and eyes, both being turned in the same direction. This phenomenon is termed by Prévost conjugate deviation (*déviation conjuguée*) of the eyes and head. It is generally temporary, and is said by Landouzy to be connected principally with a lesion of the lower parietal lobule (see also page 477). There is no perfectly constant relation between the lateral deviation and the half of the brain affected. The most common condition is that the eyes are directed toward the affected hemisphere, and so to a certain extent "look toward the lesion" and away from the paralyzed side of the body. The pupils present no constant peculiarities. Often they are of normal size. In other cases they are contracted, dilated, or unequal. No definite diagnostic conclusions can be drawn from them. In the worst cases the pupils will not react to light; in other cases they react, but often sluggishly.

During the deep apoplectic coma the extremities generally lie completely motionless and limp. In the worst cases reflex action is wholly suspended; but sometimes the vigorous thrust of a pin or the pinching of the skin will excite an occasional slow reflex twitch, or a motion as if to ward off the tormentor. Whether the apoplexy has caused hemiplegia at all, and if so in what place, cannot always be easily determined during the initial coma. Still, it is often to be observed, even now, that one angle of the mouth hangs down lower than the other, and that the corresponding cheek is more puffed out during expiration than is the other; that the extremities of one side are much heavier and fall much more limply than those on the opposite side of the body, and that the reflex action and defensive movements are almost absent upon one side (the paralyzed side), while they can be clearly demonstrated on the other.

In contrast to the usual laxness of the arms and legs during the apoplectic coma is the tonic rigidity sometimes seen in the extremities, particularly on the side opposite to the hemorrhage. This symptom seems to be especially, although not exclusively, connected with a bursting of the escaping blood into a lateral ventricle. It is rather exceptional for cerebral hemorrhage to be attended with general or unilateral epileptiform convulsions—a symptom which, as we have seen, is referable to irritation of the motor regions of the cortex.

It should be mentioned that in many cases of cerebral hemorrhage the urine passed after the attack has been found to contain small amounts of albumen or sugar. This symptom is generally ascribed to compression of the medulla from the effusion. There is usually retention of urine; in other cases there is involuntary micturition.

A certain number of patients never awake from the initial coma. Death may not be immediate, but they remain completely unconscious; the respirations become more rapid and irregular (sometimes of the Cheyne-Stokes character), and there is rattling in the throat, because mucus and saliva run down into it; the pulse, which was at first retarded, now becomes accelerated; the face grows paler and more and more sunken; the eyes are deep in their sockets; the cornea becomes opaque; and at last, after the coma has lasted some hours, or even one or two days, death occurs, often attended by a considerable rise in temperature.

This termination is, however, by no means the usual one. More frequently the patient survives the attack. The bleeding ceases, the clot contracts, and begins to be disintegrated and absorbed. At the same time the pressure exerted upon surrounding parts grows less and less, the more distant parts of the brain gradually recover from the shock, and consciousness slowly returns. The patient begins to open his eyes when he is spoken to in a loud tone; he raises his hand to his head, sighs, and yawns; gradually the intellect clears up, he tries to talk, or to express himself by signs; memory returns, and he recognizes those about him once more. Exceptionally, recovery is interrupted by a fresh and perhaps a fatal relapse. This may result from a renewal of the hemorrhage. Generally, however, improvement persists, the patient fully regains his consciousness at the end of a few days, and it now becomes possible for the first time to "estimate the damage."

The symptoms thus far described belong to severe apoplectic attacks. There are also, as we have said, cases of all degrees of mildness as regards the first onset. In these there is no deep and persistent coma. The patient loses consciousness only temporarily, if at all. He is seized with vertigo, or with sudden headache, and is for a time stupefied. Nausea and vomiting are of frequent occurrence, just as in ordinary syncope. Yet cases presenting these comparatively slight early symptoms, and with few even of these, may exhibit the genuine focal symptoms referable to the hemorrhage, such as hemiplegia, in all their severity. These latter phenomena must now be considered.

We term the symptoms of loss of function directly dependent upon the destruction of a definite portion of the brain the direct focal symptoms of cerebral hemorrhage. Where the hemorrhage takes place we have seen that a larger or smaller extent of the brain substance is completely destroyed by the sudden and forcible escape of the blood. The dimensions of this lesion are represented later by the apoplectic scar or cyst, and its position by the nature and extent of the persistent, and for the most part irreparable, loss of function. But there are, in addition to these direct symptoms, other indirect focal symptoms which outlast the apoplectic shock, and vary with the locality of the hemorrhage. These do not, however, correspond to the territory actually destroyed. They are due to the influence exerted for a certain length of time by the hemorrhagic focus upon the immediate surrounding structures. The pressure of the effusion, the disturbance of circulation resulting from it, the collateral œdema, and perhaps also the imbibition of the soluble products of disintegration of the apoplectic focus, are the chief factors in exciting these indirect symptoms. They do, indeed, outlast the initial shock, but are nevertheless temporary, vanishing sooner or later, at the end of several days or weeks, or even months. If the apoplectic shock is over, and if the symptoms still exhibited have been minutely determined, yet we are unable to say, at first, whether the existing focal symptoms are direct or indirect. We can decide about this only after further observation. If the early symptoms disappear within the next few days or weeks, or after the first two or three months, we can then affirm, retrospectively, that they were indirect. Such as outlast the first six months are to be regarded as direct, and as not destined to improve much. From a practical point of view this distinction is of extreme importance. We shall revert to the subject when considering the course of cerebral apoplexy.

A minute description of all the focal symptoms which might occur after cerebral hemorrhage, and of the light thus thrown upon the location of the hemorrhage, need not be attempted here, for it would necessitate a repetition of all the facts enumerated in the preceding chapter. It is only requisite to describe, in detail, the chief and by far the most frequent results of a cerebral hemorrhage—ordinary cerebral hemiplegia (see Fig. 207).

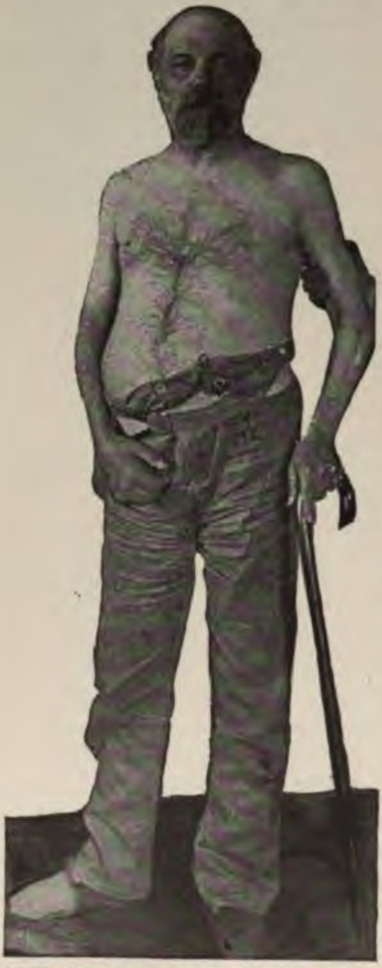

FIG. 207.—Patient with right hemiplegia. (Personal observation.)

Cerebral Hemiplegia.—It has been mentioned that most of these hemorrhages occur near the lateral ventricles. Hence, in a majority of instances, the motor pyramidal tract, as it traverses the internal capsule, is either directly destroyed or at least indirectly affected. Consequently, most patients who survive the apoplectic shock present a paralysis of that half of the body which is opposite the seat of hemorrhage. On minute examination we usually find, in the first place, that even in the distribution of the facial nerves there is a distinct difference between the two sides, the lower division of the facial (which supplies the muscles of the cheek, nose, and mouth) being evidently paralyzed on one side, while its upper division (going to the eyes and forehead) is entirely, or almost entirely, intact. The forehead can be wrinkled on one side as well as on the other, or only a little less on the paralyzed side than on the sound side; but if the patient tries to dilate his nostrils, or alter the shape of his mouth, or whistle, or blow, or talk, etc., the paralysis becomes evident. Often, indeed, while the face is quiet, it can be noticed that one nasolabial fold is obliterated, or that one corner of the mouth droops. It is an interesting fact that the paresis of the lower division of the facial is much more noticeable during voluntary efforts, as in showing the teeth, than when the patient smiles involuntarily. A patient will sometimes try in vain to draw back the corner of his mouth, then begin to laugh at his own awkwardness, and thereupon open his mouth in an almost perfectly normal manner. We have seen, in the chapter on cerebral localization, that this condition can probably be explained by the relation of the optic thalamus to the movements of mimetic expression. The difference in the behavior of the upper and the lower part of the face in cerebral hemiplegia is probably connected with the fact that the muscles supplied by the upper division (frontalis, corrugator supercilii, and to a certain extent the orbicularis) are very seldom exercised upon one side alone, but always bilaterally. Perhaps both sides receive nervous fibers from each cerebral hemisphere, so that, if a single center be intact, it alone answers for the muscles on both sides.[1] In the distribution of the lower division of the facial, also, ordinary cases of cerebral hemiplegia present almost always a more or less marked paresis, and only exceptionally a complete paralysis.

There is quite often a slight impairment of the hypoglossus in addition to the paresis of the facial nerve. If the patient puts out his tongue, its tip deviates toward the paralyzed side. This is a result of paresis of one of the genio-hyoglossi. When both these muscles contract, they may be said to push the tongue forward. If this thrust be more vigorous on one (the healthy) side, the tongue is deflected toward the other (paralyzed) side. In ordinary cerebral hemiplegia this is almost always the sole way in which the movements of the tongue can be seen to be impaired. Sometimes, however, the slight paresis of half of the tongue, combined with the facial paresis, entails a noticeable difficulty in articulation. This, to be sure, is only exceptionally great, and is often appreciated by the patient alone, who is conscious that an effort is required in order to speak.

The soft palate is rarely much affected. It may, however, hang rather lower down on the paralyzed than on the affected side, and move less. The uvula is inclined sometimes toward the healthy and sometimes toward the paralyzed side. There are no special disturbances of function as a result of these changes.

The trapezius is the only muscle of the trunk which is ordinarily much affected in cerebral hemiplegia. As a result of the paresis of this muscle, the shoulder sags, and it cannot be raised as high as on the normal side. If the patient tries to take a very deep breath, it is in some cases possible to see that the paralyzed side lags behind a little in respiration, which is due to a paresis of the respiratory muscles on that side. It is perhaps due to this that the pulmonary diseases which attack hemiplegic patients frequently develop in the lung on the affected side, where respiration is deficient.

The most important element in the hemiplegia is the paralysis of the extremities. Immediately after the hemorrhage it is often so complete that even the slightest voluntary motion in the affected arm and leg is impossible. Other cases, however, exhibit only a more or less severe paresis (hemiparesis) from the first; or the complete paralysis at any rate is confined to certain groups of muscles, the others still retaining vestiges of their normal contractility. Even when there is total hemiplegia at first some of the muscles almost always regain a certain amount of their old power later on (*vide infra*).

The behavior of the reflexes is comparatively constant in nearly all cases of cerebral hemiplegia. In general the rule is that the tendon reflexes are increased on the paralyzed side, but that certain skin reflexes are diminished.

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[1] There is a remarkable general rule which should be mentioned here, and which is perhaps to be explained in the same way—namely, that those muscles which are usually called into play in pairs, and which accordingly are probably innervated by both hemispheres, are never completely paralyzed in cerebral hemiplegia. Furthermore, it is impossible for us voluntarily to contract most of these muscles, which, as a rule, are simultaneously innervated from both sides, singly, on one side alone, or at least not without special practice; this applies, for instance, to the corrugator supercilii, the frontalis, the motores oculi, and the muscles of mastication and respiration.

The increase of the tendon reflexes is an almost constant symptom. Only when the symptoms of the initial shock are very severe, there may at first be no tendon reflexes whatever. In all cases of any duration they are, with few exceptions, exaggerated, and usually very much so. In the arm we find very lively periosteal reflexes on tapping the lower end of the radius and the ulna (see page 211); also on striking the tendons of the biceps and triceps, the clavicle, etc. In the leg the increase of the patellar reflex is most noticeable, but there is often a lively Achilles reflex also, an increase of the adductor reflexes, etc. This increase of the tendon reflexes is by no means limited to the affected side; it can almost always be detected on the sound side also, although to a lesser degree. Many have expressed the opinion that the increased tendon reflex upon the paralyzed side is a result of the secondary degeneration of the pyramidal tracts in the spinal cord. In our judgment this view is entirely unfounded, inasmuch as the exaggeration of the tendon reflex often appears within a few days, or even a few hours, after the apoplectic attack—that is, before a secondary degeneration in the spinal cord is to be thought of. We would rather seek to explain the phenomenon as being due to the suspension of certain reflex inhibitory influences because of the cerebral lesion.

There is very often found, particularly in cases of long standing affected with well-marked contractures, an increase of "direct mechanical excitability" in the paralyzed muscles, so that a tap upon them causes them to contract vigorously. We are of the opinion that a part of these contractions are of reflex origin and are due to the mechanical irritation of the fascia of the muscles (fascial reflex). There is also the direct mechanical irritation of the nerves of the muscles.

The skin reflexes in hemiplegia behave differently from the tendon reflexes. In the paralyzed arm it is usually impossible to excite any skin reflex whatever, and in the corresponding leg the reflexes produced by pricking the soles of the feet with needles are occasionally approximately equal; at other times, however, weaker than on the sound side. The change in the reflex of the big toe is very striking. Stroking of the sole of the foot generally produces a dorsal extension on the paralyzed side (Babinski), and on the well side, on the other hand, a plantar flexion (*cf.* page 207). Oppenheim's leg reflex is not as common as the Babinski toe reflex, but nevertheless it is frequently very characteristic. If the internal surface of the tibia is stroked downward with considerable force with the thumb, dorsal extension of the toes [particularly the great toe—K.], and especially dorsal extension of the foot through contraction of the tibialis anticus muscle, will occur on the paralyzed side. The abdominal and cremasteric reflexes show important changes (*vide* page 208). These reflexes are almost always much diminished or wholly absent on the paralyzed side, while they can be elicited on the sound side in normal strength, a distinction which is not infrequently of service in determining the seat of the hemiplegia when the patient is stupid or even quite unconscious. There is often a marked difference in the conjunctival reflex also. On the paralyzed side we can touch the eyeball with the finger or the head of a pin without causing an immediate reflex closure of the eye, as is the case on the healthy side. It is not easy to explain the striking behavior of the skin reflexes in cerebral hemiplegia, especially the peculiar contrast between them and the tendon reflexes. It is ordinarily assumed that certain inhibitory influences for the tendon reflexes are lost through the cerebral disease, while the inhibition of the skin reflexes is, on the contrary, put into a state of irritation. This explanation is evidently forced and unsatisfactory, and thus the suspicion is not wholly unwarranted that these skin reflexes, particularly the abdominal and cremasteric reflexes, contrary to the physiological theories thus far generally accepted, have their reflex arcs not in the spinal cord, but much higher up in the brain. The absence of these reflexes on the paralyzed side in cerebral hemiplegia is accordingly due to a direct injury of the reflex path. Of course it must be emphasized that further studies upon this important point are extremely necessary.

Sensation is but little impaired in most cases of cerebral hemiplegia, but on careful examination we often find a decided blunting of sensibility for simple contact, although the sensibility to pain and temperature and the muscular sense are retained. In isolated cases the muscular sense may be also perceptibly disturbed. [This is usually the case.—K.] Slight paræsthesia is not infrequently complained of on the affected side, especially at first. Pain in the paralyzed parts is not uncommon. It is often to be referred to special peripheral conditions (contractures, etc.), but sometimes there is apparently an irritation of the central sensory tracts (Edinger). Any marked disturbance of sensation indicates, as has been seen (*cf.* page 490), that the posterior extremity of the internal capsule is involved. Such cases are rare. In them we may observe a complete cerebral hemianæsthesia combined with the hemiplegia. According to Gowers, a temporary hemianopsia is often present directly after the occurrence of a cerebral hemorrhage. We have seen the initial hemianopsia combined with associated paralysis of the ocular muscles. It is also not uncommon to find hemiplegia associated with permanent hemianopsia, but as yet little is known about the pathological anatomy of such cases. The most likely thing would seem to be some lesion of the fibers of the optic nerve in the internal capsule or in the pulvinar of the optic thalamus. [The stereognostic sense (*vide* page 151) is not infrequently affected.—K.]

Turning now to the further course of hemiplegia, we find a new group of important symptoms. First of all should be considered the changes in the paralyzed muscles. If the hemiplegia be incomplete, even from the start, almost the normal degree of motility may in a comparatively brief period be regained by the affected side. At most there will persist a certain slight amount of weakness and stiffness, and even this will gradually diminish. From what has been already said, it is evident that in these cases the initial paresis is an indirect focal symptom, and accordingly vanishes as soon as the effusion ceases to affect the tissues not immediately involved.

Even where there is a complete hemiplegia it is exceptional for this condition to remain unabated throughout the entire region affected. After a few weeks, or even a few days, one part and another of the paralyzed half of the body begin to recover their former motility. The improvement goes on slowly, and in the most favorable cases the paralysis may for the most part have vanished at the end of some months. Usually, however, the improvement advances only to a certain point, and the condition then attained remains stationary. We now find the ordinary characteristic typical "hemiplegic," who can be recognized at the first glance by the practiced eye. The most noteworthy phenomenon in such cases is this, that, in spite of many individual differences in the final recovery of certain movements and also in the permanence of the paralysis of other groups of muscles, there is still a very striking regularity and uniformity. There is one chief fact which is very noteworthy—that almost invariably the leg regains greater power of movement than the arm. In regard to the condition of the individual groups of muscles, moreover, we almost always find the same manifestations. As has been shown chiefly through the interesting investigations of Wernicke and L. Mann, the permanent paralysis in cerebral hemiplegia, or the return of motion, never relates to individual muscles, but always to whole groups of muscles whose functions are closely connected. In the upper extremity there is usually permanent paralysis of all the muscles that rotate the arm outward (supinators of the forearm, outward rotation of the upper arm by the infraspinatus and teres minor, and fixation and outward rotation of the shoulder girdle by the lower portion of the trapezius and the rhomboidei), and of those that raise the upper arm (deltoid, serratus), and also paralysis of the movements at the elbow, especially of extension, of opening the hand (extensors of the fingers, fixation of the wrist by the flexors), and of opposition of the thumb (opponens, abductor brevis). On the other hand, inward rotation of the arm and closure of the hand are, as a rule, preserved. The patient can therefore not take hold of an object on account of the difficulty of opening the hand, but he can hold firmly what he has once grasped. Fine movements of the hand, however, are much impaired on account of the disturbances in the movement of the thumb. In the leg the flexors of the lower leg and the dorsal extensors of the foot usually remain permanently paretic, and also the abductors and inward rotators of the hip, while the iliopsoas, the quadriceps extensor, and the plantar flexors of the foot often regain their power of motion. The return of mobility in the iliopsoas and quadriceps is therefore of great practical significance, because by it the patient is enabled to walk again. Of course the patient needs the help of a cane, and the gait remains slow and labored, but the advantage of independent locomotion is extremely great. The gait of the hemiplegic is very characteristic, and is always of the same type. The affected leg has the toe turned slightly outward, it is moved forward slowly, dragging on the ground, in a slight curve whose convexity is outward. We cannot state with certainty the reason for these facts just mentioned—that both the improvement and the permanent paralysis almost always involve regularly the same definite groups of muscles. The muscles which regain the power of motion are probably those which can be most easily innervated from the healthy hemisphere on the same side of the body. There are some reasons that justify the assumption that, after the interruption of the pyramidal tracts, the subcortical motor tracts (from the optic thalamus and from the corpora quadrigemina) assume the function vicariously.

It is improbable, although, of course, not impossible, that fibers when once destroyed can undergo regeneration; but the fact above mentioned is of the greatest practical consequence that hemiplegic symptoms cannot improve materially after the first six months.

The permanently paralyzed muscles become very often contractured later on. The appearances produced exhibit considerable uniformity. The arm, which suffers more from paralysis than the leg, is also usually more contracted. The fingers are almost invariably flexed; the forearm contracted in a position of pronation, and usually flexed rather than extended; and the upper arm adducted (the pectoralis major being chiefly affected). These contractures correspond exactly to the natural positions which the paralyzed arm almost always assumes if left to itself, either from gravity or as a result of any remaining power of movement. The contractures are almost always found in the same groups of muscles which suffer relatively the least loss of power in hemiplegia (*vide supra*). Since the position once acquired from the paralysis of the antagonistic muscles cannot be altered, so-called passive contractures appear in the permanently shortened muscles. The correctness of this theory is favored by the fact that the deformity can, to a certain extent, be prevented by the persistent use of passive motion, which renders any permanent shortening of the muscle impossible. Nevertheless, Charcot and his pupils, including Bouchard, hold a different opinion concerning the origin of these contractures. They regard them as the sequel of the secondary degeneration of the pyramidal tract. This assumption is, however, highly improbable, since, in the first place, the degenerating fibers have lost the power of being stimulated, and can therefore no longer conduct any stimulus; and, moreover, the contractures do not develop in the paralyzed muscles, but principally in those which can still be moved. Contractures and secondary degeneration are consequently concomitant sequelæ of disease of the brain.

If the contractures become marked in the lower limb, they may involve either the extensors or flexors, which depends in the main upon accidents of position, etc. At the ankle we almost always find a contracture of the muscles of the calf, because the dorsal extensors of the foot are paralyzed and the foot falls into a position of plantar flexion from its own weight; and, moreover, because there is generally more mobility preserved in the plantar flexors than in the dorsal extensors of the foot. The fact that the muscular tonus is increased on the paralyzed side (analogous to the increase of the tendon reflexes) is a circumstance which is not wholly without influence on the strength of the contracture. We may mention here the fact brought forward by Hitzig that many contractures are slight in the morning, when the patient wakes from sleep, and become more marked after he has begun to move.

Among other symptoms we will mention first the interesting associated movements, which are of frequent occurrence in hemiplegics. The explanation of them is partly that the patient can no longer innervate the different paretic groups of muscles with ease. In order to do so, it is always necessary to make a forcible associated movement of the leg, when the patient is endeavoring with all his energy to move his arm, and *vice versa*. If the patient tries to raise the arm at the shoulder, he flexes his forearm at the same time. The common associated movement in the foot (dorsal extension from contraction of the tibialis anticus) on flexing the hip and knee, which was first described by me, is especially striking. Not infrequently the patient is quite unable to extend the foot dorsally as an isolated movement, but, if he draws the whole leg up to the trunk, there is then always a simultaneous marked dorsal extension of the foot with marked contraction of the tendon of the tibialis anticus. This symptom, which has been described by me under the name of tibialis phenomenon, depends probably on an interruption of the pyramidal tracts. The patients can now simultaneously ("synergically") contract only large groups of muscles, and have lost the power of distributing and restricting the contraction to single muscles. On extending the leg, we frequently see a coincident marked dorsal extension of the big toe (toe phenomenon). Similar abnormal synergic muscular movements are also observed in the upper extremity (dorsal extension of the wrist on shutting the hand, etc.). Associated movements also occur on the sound side on movements of the affected side, and it is said that the opposite condition has also been observed.

Another peculiar phenomenon must be mentioned here. It is what Weir Mitchell has termed post-hemiplegic chorea. Some time after the paralysis begins the parts affected by it exhibit involuntary movements, reminding one of chorea or athetosis (*vide* pages 203 and 490). Sometimes these movements are continuous, sometimes they occur only as associated movements in connection with voluntary motions of the paralyzed, or even of the sound, side. Hemiplegia due to cerebral hemorrhage very seldom exhibits this phenomenon. It is said to occur chiefly in focal disease of the posterior extremity of the internal capsule, and of the optic thalamus (irritation of the adjacent pyramidal tract?). It is much commoner in cerebral infantile paralysis (*vide infra*).

It is interesting to observe the trophic and vasomotor changes in the paralyzed parts. At first the skin may be somewhat redder and warmer on the paralyzed than on the sound side. Nothnagel has shown that, even in the distribution of the cervical sympathetic, symptoms of vasomotor paralysis occur. They are partly temporary and partly persistent, and comprise increase of temperature and color in the paralyzed side of the face, swelling of the eyelids, and contraction of the pupil, but they are usually slight. We very frequently find, especially on the back of the hands, more or less puffiness, which is likewise usually regarded as of vasomotor origin. It should, however, be considered that the natural movements of any part of the body greatly promote the nervous and lymphatic circulation, and that the quietude of paralysis may therefore have much to do with the œdema. In hemiplegia of some duration the extremities upon the paralyzed side are always cooler than normal, and the hand in particular is very often deeply cyanotic. The skin sometimes becomes harsh and fissured, and often is thickened. The internal surface of the hand, in case of contracture, is frequently quite wet with perspiration.

Among the specific trophic symptoms of hemiplegia Charcot includes "acute malignant bedsore." This sometimes develops with extreme rapidity within a few days after the shock, and usually occupies the buttock on the affected side. There appears a circumscribed redness with the formation of vesicles, which is soon succeeded by a deep-reaching necrosis of the soft parts. We have ourselves never met with this in a case which has been properly nursed, and we cannot therefore help thinking that its development is not due to trophic disturbance, but to pressure and to the penetration of septic matter below the skin. Of course, patients long confined to bed with hemiplegia are as liable to bedsores as are any others similarly situated.

If the hemiplegia has existed for some time, we almost always find the paralyzed muscles more or less atrophied in comparison with those on the sound side. On microscopic examination we also find an evident atrophy of the individual fibers, but this atrophy, as a rule, is not so extreme as in peripheral or poliomyelitic paralyses (see page 200). Furthermore, the atrophy is always "simple," not "degenerative," and accordingly the faradic and galvanic excitability of the paralyzed muscles is completely preserved, even if the muscles are very much atrophied. The electrical excitability does not even show a slight quantitative diminution, as careful examinations have taught us. In some cases, even in ordinary hemiplegia, the atrophy of certain muscles (for example, the interossei, the thenar muscles, the deltoid, etc.) may come on unusually early and become extremely marked, so that we may speak of a "cerebral muscular atrophy." In such cases certain trophic influences actually seem to be especially manifest. The joints of the paralytic extremities, and in particular the knee and shoulder, may exceptionally become inflamed. The arthritis is sometimes acute, and sometimes more of a chronic variety. Its cause is not evident. Charcot thinks that it is probably a neurotrophic symptom, and he is of the same mind with regard to the rarely seen swellings of the peripheral nerve trunks of the paralyzed side ("hypertrophic neuritis").

Mental symptoms are rare, except the initial loss of consciousness. There is sometimes, however, a persistent general uneasiness of mind, accompanied by great excitability and wakefulness. In a large number of cases of persistent hemiplegia there finally come on, in the course of years, constantly increasing indications of mental weakness. The patient grows dull and forgetful. Very often he exhibits a peculiar tendency to weeping, bursting into tears at the slightest provocation; but frequently he is subject to quick alternations of feeling, weeping and laughing in the same minute. The patient sometimes becomes eventually confused, excited, etc. All such pronounced conditions are usually dependent upon the general cerebral atrophy that comes from arteriosclerosis.

The general nutrition is often good for a long time. Sometimes there is even a decided tendency to corpulence. In other cases, and especially in the bedridden, marasmus gradually comes on and hastens the fatal termination, particularly if there be some intercurrent trouble, such as a bedsore or bronchitis.

We have detailed the peculiarities of hemiplegia somewhat minutely, because the statements will apply in the main to all cases of cerebral hemiplegia, no matter in what place the pyramidal tract is interrupted or by what sort of a lesion. It is needless to enter into the diverse symptoms which are caused by diversity in the exact location of the hemorrhage. The hemiplegia itself is the same, whether the effusion be in the cortex, internal capsule, crus cerebri, or pons. It is easy to infer from the preceding chapter what the accessory symptoms are which would enable us to localize the trouble. We need mention here only the frequent combination of right hemiplegia and aphasia. This occurs when there is a large effusion in the left hemisphere, extending from the internal capsule to the neighborhood of the third frontal, or possibly the uppermost temporal convolution (compare page 490 *et seq.*).

Diagnosis.—The diagnosis of cerebral hemorrhage rests on the sudden onset of the apoplectic symptoms, and on the later symptoms (if there be any) of impairment of the cerebral functions. The diagnosis can scarcely ever be made with absolute certainty, for cerebral embolism may exhibit almost identical phenomena: the differential diagnosis between the two will be given in the following chapter. Occasionally there may be danger of mistaking other cerebral affections for a hemorrhage, such as meningitis and tumors. The same may be said of a suddenly developed uræmia, of diabetic coma, and of constitutional sepsis. In these cases the rapid onset of grave cerebral symptoms of a general nature, such as unconsciousness, simulates the apoplectic coma. At times the further course of the disease alone makes a diagnosis possible.

Prognosis.—The first question is, whether the patient will survive the initial shock. The answer depends upon the severity of the early symptoms. The deeper and more persistent the unconsciousness, the more deficient the respiration and pulse, the less the prospect of recovery; but we can never decide absolutely. [A steady rise of temperature or a very high temperature is also of bad omen.—K.] If the patient have withstood the first onset and be hemiplegic, then the possibility of improvement hinges on the question whether the paralysis is a direct or an indirect focal symptom. Inasmuch as there are no means of knowing about this at first, we must speak very guardedly. [The early appearance of ankle clonus or greatly exaggerated patellar reflex is apt to portend permanent paralysis with contractures.—K.] It should never be forgotten that the hemorrhage may recur. The predisposing disease of the blood vessels renders individuals who have had one stroke liable to be visited, sooner or later, by another.

Treatment.—The treatment of the apoplectic shock demands, first of all, rest in bed, with the head and shoulders elevated. To avoid bedsores it is very important to maintain cleanliness, and to watch attentively that portion of the skin which is pressed against the bed by the weight of the body. An ice bag should be put upon the head, and particularly over that side on which the hemorrhage is supposed to be. Bleeding was formerly universally practiced, but of late its usefulness is doubted. It is at most indicated only when the deep congestion of the face, the violent pulsation of the carotids, and the full, slow pulse show increased arterial tension. In such a case, if the patient seem otherwise robust, we may bleed at the commencement of an attack, in the hope of checking the flow of blood by lowering the intra-arterial pressure. In similar conditions, experience shows that the local abstraction of blood from the temples is sometimes advantageous. The bowels should be well emptied by enemata, and later on by drastic purgatives. If the respiration and pulse fail, we may try stimulants (ether, camphor).

[Horsley and Spencer have found, by experiments on animals, that if the internal carotid be tied the bleeding from the divided lenticulo-striate artery is checked. Horsley therefore suggests that in the very earliest stage of cerebral hemorrhage ligature or compression of the internal carotid may check the bleeding, and prevent the formation of a large clot and extensive destruction of the brain. This procedure has been tried with some benefit in a few cases of ingravescent hemorrhage.

Where there is much difficulty in respiration during the period of coma, relief may sometimes be obtained by turning the patient upon the side.—K.]

When the patient is safely over the shock, our resources for aiding him in the further stages of his trouble are very limited. As long as headache and symptoms of fever persist, the application of ice to the head should be kept up, and other disturbances should be treated symptomatically. For uneasiness and wakefulness, small doses of morphin or veronal are given. Treatment of the hemiplegia must be deferred for the first three or four weeks, until all the initial symptoms of irritation are over. Then electricity plays the chief rôle. Local galvanization should be tried, the current being made to pass transversely through the head, with as much regard as possible to the position of the hemorrhagic focus; the current should be weak, and the application should occupy two or three minutes. With this may be combined galvanization of the sympathetic nerve on the side of the hemorrhage; nor should galvanization (stroking with the cathode) and faradization of the paralyzed muscles and nerves be neglected. If favorable changes take place, it is, however, uncertain how much should be ascribed to treatment; since, as has already been stated, there is often improvement without treatment.

Passive movements and massage of the paralyzed limbs are very important as a prophylaxis against contractures. Massage, and later on systematic and appropriate gymnastic exercises, may contribute much to the restoration of voluntary motions. Rubbing with spirits of camphor, chloroform liniment, spirits of mustard, etc., which are much employed, have a favorable influence through the massage and cutaneous stimulation.

Internally, potassium iodid is frequently given, out of regard for its "absorbent" properties. We may also try the effect of strychnin; it is most adapted for cases of some duration. [Strychnin should be prescribed very cautiously in cases where the blood pressure is high and in cases where there is any spasticity, as it has a tendency to aggravate spastic phenomena.—K.]

As to baths, they should not be too warm—that is, not over 90° to 93° F. (33° to 34° C.). Moderately warm baths, medicated with common salt if it seems desirable, and employed three or four times a week, or artificial carbonic-acid baths, frequently seem to be beneficial. If it be thought best to send the patient to a regular bathing place, Wildbad, Ragatz, Teplitz, Wiesbaden, Oeynhausen, Nauheim, etc., may be chosen. At all these places, too hot baths should be avoided.

Hemiplegic paralysis often lasts so long that the physician must repeatedly change the details of treatment, so as to support the courage and patience of the sufferer. Particular care should be given to regimen, in order that any recurrence of the hemorrhage may, if possible, be avoided. The diet should be simple; any large amount of alcohol should be forbidden, and there should be no severe bodily exertion or mental excitement.

CHAPTER IV

CEREBRAL EMBOLISM AND THROMBOSIS (ENCEPHALOMALACIA)

(Softening of the Brain from Embolism or Thrombosis)

Ætiology and Pathology.—Occlusion of the cerebral arteries is one of the most frequent injuries inflicted by embolism. Usually the emboli originate in the left side of the heart, from thrombi in the left auricle, or from the thrombotic deposits which form upon the valves of the left ventricle in chronic endocarditis (mitral or aortic disease). Chronic arteriosclerosis may also lead to thrombosis in the larger arteries, particularly the aorta; and in case the cerebral arteries themselves are extensively atheromatous, the larger vessels at the base of the brain may furnish material for embolism of the smaller cerebral arteries.

Thrombosis of the arteries of the brain is always due to primary disease of the blood vessels, the most common cause being the chronic arteriosclerosis just mentioned. Wherever the atheroma has altered the normal structure of the intima, deposits of fibrin may take place. Their development is further promoted by the subnormal elasticity of the arteries, and by the occasional narrowing of their lumen; for thus the flow of blood is rendered slow, if not completely checked. It is easy to understand that thrombosis and embolism may each give rise to the other. From every thrombus an embolus may be detached; and every firmly lodged embolus may form a nucleus for thrombosis.

Next to arteriosclerosis the most frequent cause of a cerebral thrombus is syphilitic endarteritis. In the chapter on cerebral syphilis this subject will be more fully considered. Whether thrombi ever form here independently of disease of the vessels is doubtful. An apparently spontaneous thrombosis is now and then seen in patients who are cachectic or severely ill (cancer, severe typhoid fever, etc.). Such cases are explained either by the existing cardiac weakness, or perhaps by an abnormal tendency of the blood to coagulate.

In whatever part of the arterial system complete occlusion has been produced by an embolus or thrombus, the results depend upon the possibility or impossibility of blood reaching by collateral channels the region thus deprived of its ordinary supply. If the collateral circulation prove efficient, no harm is done; if not, the tissues must perish and undergo "softening." It is thus a matter of the greatest practical import that the arteries of the brain stem, and particularly the branches of the middle cerebral artery in the fissure of Sylvius, which supply the great central ganglia and internal capsule, are all "terminal," in Cohnheim's sense—that is, they form few anastomoses with neighboring vessels. Now, the middle cerebral artery and its branches are known from experience to be peculiarly liable to embolism, above other cerebral arteries. Hence we see why the region they supply suffers so severely and so very frequently from emboli. It is noteworthy that the left middle cerebral artery is more frequently plugged by emboli than is the right. In the centrum ovale and cortex there is more opportunity for collateral compensation than in the central ganglia; but even here the supply of blood often proves insufficient, as is shown by the not infrequent occurrence of spots of softening in the cortex and the white substance of the cerebrum. On the other hand, embolic foci are much rarer in the crura cerebri, pons, and cerebellum.

The various steps in the process which begins with embolic or thrombotic occlusion and ends in softening of the brain substance are essentially the same as occur in other organs (compare chapter on Pulmonary Embolism, Vol. I, page 332). The tissues which are deprived of their arterial blood perish, become disintegrated, and are transformed into a soft homogeneous mass. Into the empty portion of the artery, beyond the embolus, blood flows in the reversed direction from the veins, and, if the anatomical relations permit, from the minute arteries in the neighborhood; but the supply is not sufficient to nourish the tissues. The walls of the blood vessels become abnormally permeable and delicate, so that some red globules invade the disintegrating region by diapedesis, and others in the way of minute but genuine ecchymoses. Actual infarctions, are, however, never formed in the brain, perhaps because, as Weigert suggests, the nervous structures swell so much as to exclude any large amount of blood; but the little punctiform ecchymoses are in many instances so abundant that the whole softened spot seems distinctly reddish or yellowish. This red or yellow softening is also due in part to the tissues being stained by the dissolved pigment of disintegrated blood globules. If the discoloration be not very striking, then we speak of a white softening.

Fresh foci of softening are seen through the microscope to be composed of drops of myelin, bits of swollen nerve fibers, numerous fatty granular cells, and free fat globules. The time required for these changes is one or two days. If within that period a compensatory collateral circulation is set up, the nervous structures may be restored and resume their functional activity. If not, the tissues perish and become disintegrated. The white blood corpuscles and leucocytes (and possibly also the endothelial cells of the blood vessels, and the neuroglia and ganglion cells) absorb the fatty detritus thus made, and come to form the fatty granular cells above mentioned. If the patient live, the dead and disintegrated tissue is gradually absorbed, and it may even finally be replaced by a cyst not differing in appearance from those which occur after cerebral hemorrhage. A minute focus of softening may also be replaced at times by indurated cicatricial tissue.

If portions of the surface of the brain become softened, quite a deep depression often results. This is filled up in part with serous fluid and in part by hyperplasia of the pia mater. Sometimes the convolutions are still recognizable in places; but they are atrophied, of a yellowish color, and of a greatly increased consistency, due to the growth of cicatricial tissue.

Clinical History.—The occurrence of cerebral embolism is attended with almost precisely the same sort of shock as is cerebral hemorrhage. We do not need to enter into the particulars again here, but will refer to the preceding chapter (*vide* page 500). In embolism, also, the intensity of the shock varies from transitory confusion of intellect or slight vertigo to the deepest persistent coma. One chief factor in determining the nature of the case is the size of the occluded artery; another is its position, according as the embolism has taken place in the hemispheres or toward the base of the brain. In general, however, the shock of embolism, especially the loss of consciousness, is not often so severe and long continued as that of hemorrhage; and embolism does not so frequently give rise to symptoms of cerebral compression, including slowing of the pulse. The temperature does not usually show the decline which is seen in fresh cerebral hemorrhage, but moderate fever sets in, as a rule, in the course of a few days. On the other hand, epileptiform convulsions are seen more often in embolism than in hemorrhage. Embolism again may have a rather slow onset where there is at first a small embolus, and this becomes the nucleus of a gradually formed thrombus.

It is not easy to explain why there should be a shock at all in case of embolism. Perhaps the main reason is the diminution of pressure which the embolism occasions in the region directly affected and in its neighborhood. The portion of the artery beyond the plug becomes empty and not only causes a draught upon the blood and lymph, but subjects all the soft surrounding structures to a diminution of tension and to a certain amount of strain (Wernicke). It is not impossible, however, that the disturbance of circulation occasioned in the surrounding blood vessels, including the capillaries of the cortex, by sudden embolism of a good-sized artery is of itself enough to account for the symptoms of shock.

With regard, also, to the persistent symptoms of embolism we may be equally brief, inasmuch as they very closely resemble those which follow a cerebral hemorrhage. As has been said, it is only when a compensatory collateral circulation is developed within the first forty-eight hours that the early symptoms of focal disturbance can entirely vanish. After this period, the tissues which have been deprived of their normal blood supply must undergo necrosis; although there is still room for hope that some of the symptoms will prove indirect, and therefore capable of improvement, so that embolic hemiplegia, like that from hemorrhage, may improve decidedly in the course of the first few weeks.

Inasmuch as the middle cerebral artery is by far the most frequent seat of cerebral embolism, and inasmuch as this artery supplies the internal capsule as well as the great central ganglia, the most frequent focal symptom of embolism of the brain is ordinary cerebral hemiplegia, all the features of which have been depicted in the preceding chapter. Aphasia is associated with it with comparative frequency, for, as already mentioned, the left middle cerebral artery is especially apt to suffer. Less frequent is paralysis of a single member, due to cortical embolism, or an occipital lesion with hemianopsia.

Where softening is the result of thrombosis, the symptoms are but seldom abrupt in their onset. Usually the focal symptoms and the more general ones (such as unconsciousness) are developed rather gradually. This is most often seen in so-called senile softening of the brain, a disorder almost always caused by sclerosis of the cerebral arteries. The various symptoms generally come on under cover of repeated relapses and fresh aggravations of the disease. A severe initial shock seldom occurs; but almost invariably there is a gradual and progressive dementia.

The further course and the final termination of cerebral softening vary as do those of cerebral hemorrhage. Embolism of a larger artery may cause speedy death. If, however, the first shock passes away, the impairment of function which may be left behind may last for years without seriously affecting the general health. There is always danger of a recurrence when the source of the embolism continues unchanged, as in cardiac disease or atheroma.

Diagnosis.—Both the onset and the persistent focal symptoms are so much alike in hemorrhage and embolism that in many cases it is utterly impossible to decide which caused the apoplexy and hemiplegia. The following are factors in making a differential diagnosis, if one can be made: (1) It is very important to find whether there be any source for an embolus. Thus, if the patient have valvular cardiac disease (particularly mitral), embolism is more probable than hemorrhage. (2) A young person is, on the whole, more apt to have embolism than hemorrhage. At a later period of life one is about as probable as the other. (3) When the shock is severe and persistent, with red face, strong pulsation of the carotids, and signs of cerebral compression (slow pulse), we should think of hemorrhage rather than embolism, in which latter the initial symptoms are sometimes comparatively slight (*vide supra*). (4) Finally, it is sometimes possible to obtain support for a diagnosis of cerebral embolism by demonstrating embolism elsewhere, as in the fundus oculi, by means of the ophthalmoscope.

In rare instances, also, tumors of the brain, into the substance of which hemorrhage takes place, may induce symptoms closely simulating a primary apoplectic shock, as may also abscesses which have been previously latent, and then suddenly burst into a ventricle. In such cases a correct diagnosis is seldom possible.

Softening due to a thrombus is most readily diagnosticated in those cases where there is cerebral syphilis (*q. v.*). Senile softening is inferred from the age of the patient, the signs of general arteriosclerosis, the abrupt advances of the disease from mildness toward severity, and the developing dementia.

For the prognosis and treatment of cerebral embolism we may refer simply to what was said in the preceding chapter.

APPENDIX

ARTERIOSCLEROTIC BRAIN DISEASE

In the two preceding chapters on hemorrhage and embolic softening of the brain, we have repeatedly emphasized the great significance of arteriosclerosis in the ætiology of these important pathological lesions. But even if a distinct apoplectic attack or a pronounced hemiplegia does not ensue, extensive arteriosclerotic changes in the cerebral vessels frequently produce a distinctive clinical picture which we must briefly review here.

As a rule, this disease occurs in older people, in whom arteriosclerosis is also causing symptoms in other vascular territories of the body (heart, kidneys, extremities). However, the cerebral symptoms may occasionally occur almost independently. In the case of comparatively youthful individuals, in whom arteriosclerosis occurs remarkably early, special ætiological factors (alcohol, nicotin, heredity, gout, etc.) must be considered.

The disease of the cerebral vessels causes an obstruction of the circulation, and a consequent insufficient nourishment of the brain tissues. The first and most general consequence of this is a diminution in the general cerebral functional activity. Consequently, this disease, at first, frequently manifests itself only in the form of greater bodily and mental fatigue, general mental weakness, loss of memory, loss of mental activity, interest, and energy. In addition, marked insomnia occasionally exists, while other cases frequently fall asleep even during the day. Not infrequently disagreeable subjective symptoms (headache, vertigo, tinnitus aurium, etc.) are associated with these manifestations. A certain general disturbance of nutrition (emaciation) is often striking. Occasionally the motor weakness is particularly prominent, and the gait and all other movements become slower and more uncertain. The patients frequently walk with short, dragging steps, fall easily, cannot help themselves very well, etc. For very evident reasons this condition is not infrequently associated with true apoplectic attacks of a light or even of a grave sort. Isolated epileptiform attacks may also occur. To this are also frequently added symptoms in other organs (lungs, heart, etc.). [*Cf.* chapter on Senile Dementia, page 693.—K.]

The anatomical basis of all these pathological symptoms is principally cerebral atrophy, either general or restricted chiefly to certain areas of the brain. The convolutions become narrower, the sulci deeper, the entire brain becomes smaller and loses in weight. Microscopical examination shows a partial disappearance of nerve fibers and cells, changes in the smaller blood vessels, and an increase of the interstitial neuroglia. Frequently we find in advanced cases of this sort either isolated large, or numerous small, areas of softening or loss of brain substance.

The diagnosis of cerebral arteriosclerosis is, as a rule, not difficult, if we take into consideration the general conditions and the special symptom-complex. However, confusion, with paretic dementia, paralysis agitans, tumors of the brain, etc., may occur (*vide* corresponding chapters). The prognosis is evident from the character of the disease, but is uncertain in the individual case, since numerous unforeseen accidents (apoplexy, embolism, etc.) may occur. The treatment must be directed primarily toward conserving the bodily strength and mental energy in every possible way. The mode of living of these patients must therefore be accurately regulated. Residence in a quiet country or mountain resort is of decided benefit. The careful use of baths (salt baths, CO_2 baths) is frequently advantageous. Of the internal remedies, the iodin preparations and general tonics are to be principally considered; in addition, there are numerous symptomatic remedies, according to the peculiarities of the case. [The use of electrical currents of high frequency is often beneficial. They lower the blood pressure and improve the general condition.—K.]

CHAPTER V

INFLAMMATION OF THE BRAIN

(Acute and Chronic Encephalitis)

1. ABSCESS OF THE BRAIN (SUPPURATIVE ENCEPHALITIS)

Ætiology.—In most cases of cerebral abscess it is possible to demonstrate with certainty that infectious material capable of exciting suppuration has penetrated to the encephalon. This is particularly true of those not very rare abscesses which follow injuries of the scalp, cranium, or brain (traumatic abscess of the brain). Here the wound is probably almost always an open one, affording free ingress to the infection. In the rare cases of traumatic cerebral abscess, apparently without any open wound, small injuries of the skin have probably been overlooked. It is not essential that the bones should be injured, however, for experience shows that, even where the soft parts alone are wounded, suppuration may extend to the brain. The manner in which this extension takes place determines the question whether a suppurative meningitis (*q. v.*) or an abscess shall be developed. Not infrequently we find the two combined. Between the trauma and the appearance of symptoms of abscess a long interval of time may elapse (according to some reports even years). Another source of traumatic cerebral abscess is foreign bodies which penetrate the brain (for instance, through the orbit), and thus carry septic matter into the very substance of the organ.

Besides traumatic causes, any existing suppuration in neighboring parts may, by direct extension, occasion cerebral abscess. The same processes are prominent in this connection which we have already found to excite purulent meningitis (*vide* page 454)—particularly suppuration in the middle ear and in the petrous bone (chronic purulent otitis media, caries of the petrous bone). As a rule, the suppuration attacks the brain by contiguity, after the disease of the bone extends to the dura, and this becomes infected. In rare cases extradural abscesses are also formed. If the thin roof of the tympanic cavity is broken through, the abscess usually forms in the temporal lobe, while suppuration in the mastoid usually leads to cerebellar abscess. The excitants of suppuration may also reach the brain along the lymph sheaths of the acoustic and facial nerves or by means of the perivascular lymph sheaths and veins. The brain abscess then appears to be separated from the superficial abscess by a layer of apparently normal brain tissue. About a third of all abscesses of the brain are due to purulent ear disease. The danger of the development of a cerebral abscess arises especially when the discharge of pus is checked by the growth of granulations, by the formation of cholesteatoma, etc., and also in acute exacerbations of chronic purulent otitis. Abscesses of the frontal lobe, following suppurative processes in the nasal cavity and the ethmoid, are much less common.

In yet a third class of cases the morbific agents are conveyed from foci of disease situated in distant parts of the body. These are metastatic or embolic abscesses. They occur, for instance, in pyæmia and ulcerative endocarditis. Abscesses of this sort, however, are usually small, and seldom are prominent in modifying the grave general disease. Of more importance are cerebral abscesses connected with certain suppurative affections of the lungs and pleura. These are not so very rare. They are oftenest associated with fetid bronchitis, pulmonary gangrene, and empyema. Purulent meningitis (*q. v.*) may occur in the same way. There can be no doubt that virus is in some way conveyed to the brain, but just how is not yet known.

In some few cases of cerebral abscess no certain ætiology can be made out. To these the term idiopathic is applied. We met with several of them just at the time of an epidemic of cerebro-spinal meningitis, and it therefore seems reasonable to suspect that possibly many apparently spontaneous cerebral abscesses are referable to the same poison as is epidemic meningitis. In other cases we have to do with other forms of infection.

From the variety of exciting causes, it is evident that in cerebral abscess the special variety of the purulent infection is not always the same. We find streptococci or staphylococci most frequently in the pus of an abscess, or under some circumstances the pneumonia diplococci, proteus vulgaris, etc.

Pathology.—The pathological anatomy of abscess of the brain is precisely the same as that of abscesses in other organs. The size varies from minute foci hardly as large as a lentil to extensive cavities occupying nearly the whole of one lobe. The traumatic and otitic brain abscesses are usually single, but metastatic abscesses are not infrequently found in considerable numbers. The pus is usually greenish yellow. It may be odorless or offensive. Sometimes remnants of the destroyed ("melted") nervous tissue and red blood globules are mixed with it. The walls of the abscess often bulge out irregularly. The cerebral parenchyma around the abscess for a greater or less distance exhibits white softening. This softening is due partly to the pressure and partly to the spread of the inflammation. An abundance of granular cells is generally to be found in the tissues near the abscess.

If the abscess be very large and reach nearly to the surface of the brain, it may sometimes be recognized externally by a distinct bulging and perceptible fluctuation. The convolutions on the surface of the affected hemisphere are almost always flattened. If the abscess extends completely to the surface of the brain, purulent meningitis is excited. The complication of cerebral abscess with purulent phlebitis and sinus thrombosis has also been quite frequently observed. Abscesses which are centrally situated not infrequently burst into a lateral ventricle. An abscess of long standing may finally become encapsulated—that is, become encased in a smooth, firm layer of connective tissue, which prevents further extension of the process. The pus inside gradually becomes thick and cheesy. Apparently, however, it is very seldom entirely absorbed, and a new acute outbreak of inflammation, excited, for example, by an injury, is always among the possibilities.

Clinical History.—Small abscesses, and even large ones, may for a long time have scarcely any symptoms. This is particularly true of apparently idiopathic abscesses, and of those which develop very slowly and insidiously after apparently insignificant injuries of the head or in connection with chronic inflammation of the middle ear, etc. There is sometimes a long "period of latency" or a longer period of indefinite mild precursory symptoms (occasional headache, vertigo, general malaise, a slight elevation of temperature, etc.) preceding the onset of severe cerebral symptoms.

In abscesses following more extensive injuries, the symptoms are more violent from the start; this is also true of many abscesses which develop acutely and enlarge rapidly. The symptoms can hardly be distinguished from acute meningitis. The patient is dull, or grows delirious; he has violent headache, vomiting, symptoms of motor irritation (general or limited convulsions), and fever. Sometimes the temperature repeatedly rises to a high point. Definite focal symptoms (hemiplegia or monoplegia, partial cortical epilepsy, aphasic disturbances, hemianopsia, etc.) may manifest themselves according to the special situation of the abscess. The loss of consciousness becomes more and more complete, and in a comparatively brief time (one or two weeks) there may be profound coma and death. Rarely the violent symptoms abate, and the acute passes into a chronic stage.

The symptoms of cerebral abscess, when it pursues a chronic course, may be divided into (1) the general symptoms and (2) the focal symptoms, resulting from the special position of the abscess. There is no other localized cerebral disorder in which the focal symptoms are so often absent, either for a long time or even throughout the illness. This is partly because the abscesses are often situated in parts of the brain, injury to which does not occasion any demonstrable focal symptoms. Such localities are the white matter of the frontal lobe, the hemispheres of the cerebellum, etc. A second reason is that an abscess is not very apt to excite indirect focal symptoms by its influence upon the parts around it.

Among the general symptoms, the most important is a persistent, deeply situated, dull headache. For a long time it may be the only symptom, especially when the abscess is gradually developed after an injury to the head or after aural disease of long standing. The pain is referred mainly to the seat of the abscess, but the exceptions to this rule are not infrequent. The skull is sometimes especially sensitive to percussion in the vicinity of the abscess, but we should not lay too much stress upon this symptom. Another frequent symptom besides headache is vertigo; and there may also be vomiting, either after meals or entirely independently of food. Another valuable symptom in diagnosis is the irregular fever, sometimes slight, and sometimes exhibiting great elevations with intervals between them; but in many cases also, particularly where the abscess is encapsulated, there may be no fever whatever. The pulse is often slow. An important diagnostic point is that choked disk and optic neuritis may occur in abscess of the brain, but they develop far less frequently, and only exceptionally do they reach so high a degree as in tumors of the brain (*q. v.*).

The general health may be but slightly disturbed. Usually, however, there is decided indisposition. The patient is pale, has no appetite, loses flesh, and sometimes shows slight mental changes (a melancholy condition, temporary confusion, etc.).

As to the focal symptoms, little need here be added to what is contained in Chapter II of this section. Abscesses involving the motor region of the cortex have been repeatedly found to cause limited epileptiform attacks and monoplegic paralysis. It is particularly characteristic that, as the abscess grows, one symptom of paralysis is added to another, and that often the advance of the paralysis is ushered in by epileptiform convulsions. Abscesses in the occipital lobe have repeatedly been observed to cause hemianopsia, and abscesses in the temporal lobe the different forms of sensory aphasia (word deafness, verbal amnesia, paraphasia, etc.), and these facts have been used in making a diagnosis of the location of abscesses. Abscesses in the cerebellum may remain unsuspected for a long while, but in other cases characteristic cerebellar symptoms (staggering gait, vertigo, occipital headache, cervical rigidity) may be evident, as well as severe general symptoms.

The duration of chronic cerebral abscess varies greatly. In most cases an abscess lasts for months, and sometimes it has certainly lasted for years. Particularly when the abscess gives rise to no symptoms, or merely to slight and indefinite ones referable to the head, it may last a very long time. It is quite often the case that there are separate attacks of the severe symptoms, such as headache, vomiting, and fever, and that in the intervals of variable duration between these paroxysms the patient feels pretty well.

The final termination of cerebral abscess, unless there be surgical intervention, is almost always fatal. Recovery is not impossible, but it has been seen with certainty in only a very few instances. Death either comes on gradually, when, as the abscess grows larger, all the symptoms become correspondingly aggravated, or it may happen quite suddenly. Sometimes it is brought about by the abscess bursting into a lateral ventricle, or by the supervention of meningitis. Often a cerebral abscess terminates in a sudden and unexpected death, where no immediate cause for the event can be found.

Diagnosis.—Although it is often possible to make a correct diagnosis of cerebral abscess, yet there is usually a good deal of difficulty in arriving at such a conclusion, and entire certainty is seldom attainable. The most important points in diagnosis are: (1) The demonstration of some cause, such as trauma, chronic otitis, fetid pulmonary diseases, or empyema. (2) The presence of general cerebral symptoms, such as headache, vertigo, and vomiting; and the fact that these are sometimes better and sometimes worse. To aid in excluding tumor, we have (3) the febrile symptoms frequently caused by abscess, but rare in case of tumor; and (4) that a marked choked disk occurs more frequently in brain tumor than in brain abscess. (5) A pronounced leucocytosis may occasionally be of value in favor of the diagnosis of an abscess. The focal symptoms, if there be any, are not characteristic. They grow worse by fits and starts in tumor just as in abscess. One fact, however, is of value—namely, that while tumors (*vide infra*) frequently cause disturbances in the area of distribution of the nerves at the base of the brain (such as paralysis of the oculomotor), an abscess does this only exceptionally. It is often scarcely possible to make a differential diagnosis between acute abscess and purulent meningitis unless focal symptoms are developed, which indicate the existence of an abscess.

The timely recognition of an abscess of the brain following purulent otitis is of the greatest practical importance. It is important to know that cerebral symptoms (headache, vomiting, vertigo, stupor, etc.) may also occur from retention of pus in the middle ear, and that they may disappear completely after evacuation of the pus by paracentesis of the membrana tympani. If we find no retention of pus in the ear, however, we must suspect an abscess of the brain, if any persistent cerebral symptoms arise. The peculiarities of the otitic cerebral abscess will be discussed more in detail in the appendix to this chapter.

Treatment.—The only possible way in which to cure an abscess is by operation; the skull is trephined and the abscess laid open. The earlier and the more accurately the diagnosis of abscess can be made, the more favorable is the outlook for the patient. With the present antiseptic modes of procedure, the dangers of an operation are slight, and the series of successful results that have already been obtained urgently demand further surgical intervention. Many a life can be saved by timely surgical interference, especially in abscesses of the brain following chronic ear disease (*vide infra*). For particulars we must refer to special works on surgery.

If operation be not justifiable, nothing but purely symptomatic treatment is left us. Ice to the head, narcotics, potassium bromid, electricity, and sometimes also the local abstraction of blood, are the main remedies beyond general hygienic measures.

APPENDIX

PURULENT CEREBRAL INFLAMMATIONS AS A COMPLICATION OF DISEASES OF THE EAR

On account of the frequency and great practical importance of cerebral (intracranial) diseases which occur in conjunction with suppurative processes in the ear, we here give a brief summary of the most important facts bearing on this subject.

1. **Otitic Pachymeningitis.**—If a suppurative process in the middle ear has destroyed the petrous portion of the temporal bone to such an extent that the adjacent dura mater is reached, if the inflammatory process becomes en-

capsulated soon enough, a circumscribed extradural or subdural abscess is formed in many cases. Abscesses of this sort may rupture spontaneously into the middle ear, or, externally, behind the mastoid process. As a rule, however, without timely operative intervention, they go on to meningitis, brain abscess, etc. The symptoms of dural abscesses are quite vague—headaches, pain on percussion of the skull. Fever is either absent or slight.

2. **Otitic Leptomeningitis.**—This condition arises through extension of the infection by continuity, or the pyogenic bacteria are carried through the lymphatic sheaths of the acoustic or facial nerve, the small arteries, or through the veins. The clinical symptoms have been previously described in detail. We distinguish the rare mild cases of serous meningitis with a benign course and the ordinary severe cases of suppurative meningitis.

3. **Otitic Brain Abscess.**—Otitic brain abscess is caused by the transmission of pyogenic bacteria from the subarachnoid lymph spaces through the lymphatic sheaths of the blood vessels which enter the brain. Thrombosis of the affected blood vessels often gives rise at the same time to an anæmic necrosis of that portion of the brain supplied by the thrombosed blood vessel. Otitic brain abscesses are almost always located in the white matter of the brain, the most frequent sites by far being the base of the temporal lobe or the anterior half of the corresponding cerebellar hemisphere. The course of the disease is generally divided into several stages: (*a*) The initial stage of meningeal irritation, with fever, headache, vomiting, general malaise; (*b*) stage of developing cerebral pressure, with increasing headache and malaise, and attacks of vomiting, sensitiveness of the skull to percussion, pallor and loss of appetite, fever being either absent or very slight. These symptoms gradually merge into (*c*) the stage of marked focal symptoms, which are frequently combined with evident symptoms of increased general cerebral pressure (slowing of the pulse, optic neuritis or choked disk, somnolence and stupor). Since brain abscesses of this sort, as has been previously mentioned, are most frequently located in the temporal lobe or the cerebellum, particular attention should be paid to the focal symptoms of these portions of the brain. Abscesses of the left temporal lobe can be diagnosticated with the greatest comparative certainty by the discovery of aphasic disturbances, word deafness, verbal amnesia, optic aphasia, etc. (Oppenheim). If the abscess extend deeper, it may cause hemiparesis, hemianopsia, and ataxia by indirect pressure, and paresis of the oculomotor or abducens nerve by direct pressure on the base of the brain. Otitic cerebellar abscesses cause occipital pain, slight cervical rigidity, dizziness, and unsteady gait (cerebellar ataxia). Local tenderness of the skull on percussion, generally above the upper border of the auricle, should also be looked for. (*d*) If operative interference be impossible, general coma finally supervenes. Death is occasionally caused by rupture of the abscess into the ventricle, or by the development of general meningitis.

4. **Otitic Sinus Thrombosis.**—Otitic sinus thrombosis may originate as a primary disease by extension of the suppuration to the wall of a sinus, or, secondarily, by extension from a primary thrombus of a small vein of the ear. Thrombosis of the sigmoid sinus (a part of the transverse sinus) occurs most frequently in conjunction with suppuration of the mastoid process, but suppurative phlebitis of the petrosal sinus and the bulbus jugularis also occurs. The principal symptom is the pyæmic fever, combined with chills and inter-

mittent high elevations of temperature. Pyæmic metastases in the lungs, joints, kidneys, etc., frequently develop. Occasionally a painful swelling develops behind the mastoid process, and headache, general toxic-infectious symptoms and enlargement of the spleen supervene. In rare cases the thrombosed jugular vein can be felt. The prognosis without operative interference is almost invariably fatal. If, on the contrary, the sinus is opened in time, and the jugular vein is ligated, the prospects for recovery are very good (approximately sixty to seventy per cent of recoveries).

2. ACUTE AND CHRONIC NON-SUPPURATIVE ENCEPHALITIS

While the spinal cord is quite frequently affected by idiopathic circumscribed inflammation (transverse myelitis and acute poliomyelitis), analogous cerebral disease is much more exceptional. At any rate we may regard it as definitely settled that acute inflammatory, non-suppurative processes, which probably depend on some infection from without, may also occur in the brain. In the present state of our knowledge, the following forms of encephalitis may be clinically distinguished:

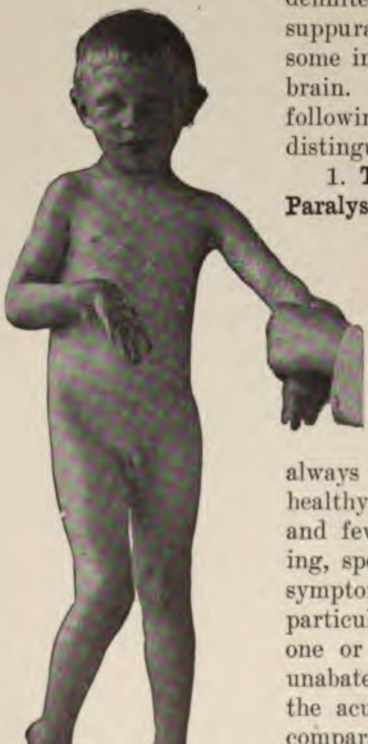

FIG. 208.—Cerebral infantile paralysis. Right hemiplegia. (Personal observation.)

1. **The Acute Encephalitis of Children. Cerebral Paralysis of Children** (*Spastic Infantile Hemiplegia of Benedikt*).—Children not infrequently suffer from a definite form of hemiplegia which, in a number of cases, at least, probably depends on an acute encephalitis, and therefore deserves a brief description in this place.

The patient is usually between one and four years of age. The symptoms almost always begin acutely. Having been previously healthy, the child is suddenly attacked with malaise and fever. Very often there is nausea and vomiting, speedily or at once followed by grave cerebral symptoms. There is stupor, and convulsions are particularly frequent. This condition may last but one or two days, or may continue, perhaps with unabated severity, for two or three weeks. Then the acute symptoms abate, and the child recovers comparatively fast; but it is noticed by the parents to be paralyzed, and this paralysis seldom disappears, although it may diminish. Sometimes the paralysis follows the convulsions, without any other definite encephalitic symptoms.

If such children come under observation after the paralysis has lasted some time, as is usually the case, their condition is generally as follows: The cranial nerves, as a rule, are but little affected. The main change is in the extremities of one side, and the arm is almost always worse than the leg. The affected parts betray an arrest of development and more or less impairment of motion; in many cases a marked exaggeration of the tendon reflexes, and almost invariably contractures of more or less severity (see Fig. 208). Further details of the hemiplegic disturbance need not be discussed here, as the conditions in general are the same as in hemiplegia of adults (*vide supra*, page 504). The muscles, although generally somewhat atrophied, never exhibit the reaction of degeneration. Sensation, as a rule, is unimpaired; but on careful examination we sometimes find in the paralyzed arm a slight diminution of the sensibility to touch and of the kinæsthetic sense, and especially a loss of the ability to recognize objects by the aid of the sense of feeling when the eyes are closed (see page 152). Permanent hemianopsia may also be associated with the hemiplegia. Symptoms of motor irritation are found upon the affected side with striking frequency, which, to a certain extent, is characteristic of infantile hemiplegia as opposed to the hemiplegia of adults; the commonest are athetoid movements (hemiathetosis, hemichorea), and associated movements are also not infrequent. The constant movements of the fingers resulting from the athetosis sometimes render the articulations so lax that it is possible to make the fingers assume an angle of ninety degrees, or even less, with the back of the hand, at the metacarpo-phalangeal joint. The child may keep making motions with its paretic arm while walking. It is not very rare for epilepsy to develop later. There are convulsive attacks, which usually begin on the paralyzed side, but which may later affect the whole body. With right hemiplegia we sometimes see coexisting disturbances and defective development of speech. The intellectual development of many such children is tolerably normal. Others, however, are more or less demented, or betray defective moral sense. [See the chapter on Idiocy, page 702.—K.]

The whole course of the disease suggests very strongly an acute encephalitis. The process is probably in most cases limited more or less completely to the motor region of the cortex, and hence it has been called "polioencephalitis."[1] It reminds one forcibly of the acute poliomyelitis of children, with a difference merely in the localization of the disturbance. It is not impossible that the two diseases have a very similar, if not identical, ætiology. They can hardly be differentiated in their initial stage; but when further developed they cannot be confounded, because the cerebral disease causes unilateral paralysis, motor irritation, leaves the electrical reactions unimpaired, and frequently causes exaggeration of the tendon reflexes. It deserves mention that precisely similar phenomena may be presented by children during recovery from measles, scarlet fever, pneumonia, and other acute infectious diseases.

The pathological conditions of the early stage have not yet been studied. Long after the process has run its course, the affected portion of the cerebrum presents marked atrophy with cicatricial contraction. If the cortex has been affected we find a cicatricial depression, a loss of substance, a so-called porencephaly. At these places the pia is thickened. Sometimes limited cystic formations are found. The pyramidal tract exhibits a secondary descending degeneration. It is thus evident that the process is, from an anatomical point of view, precisely like the atrophy of the anterior cornua occasioned by poliomyelitis.

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[1] [This name, however, was previously applied by Wernicke to that form of external ophthalmoplegia due to changes in the nerve nuclei in the pons (*vide* page 440).—K.]

Of course not all the hemiplegias arising in childhood are due to an encephalitis, for in not a few cases infantile hemiplegia may be due to embolic softening, and perhaps sometimes to hemorrhage. In some cases it is often hard to decide whether we should regard a cerebral infantile paralysis as the result of a previous encephalitis or of some other form of cerebral disease; but it seems to us highly probable that a large part at least of the very peculiar infantile hemiplegias must have their special cause, which we have assumed to be encephalitis. The chief thing to be considered in the diagnosis is the peculiar onset of the disease with fever, convulsions, etc., in a child who was previously perfectly healthy. This distinguishes encephalitic paralysis from hemiplegia existing since birth (congenital defect, injury at birth). [There is really little pathological evidence of acute polioencephalitis in children. Sachs and Osler, from the study of a large number of cases, find that the cause of the paralysis is almost always hemorrhage or embolic softening.—K.]

The treatment at first is to be governed by the same rules as in the initial period of acute poliomyelitis (*q. v.*). The hemiplegic symptoms which persist after the first few months will never improve much. The most we can do will be by means of electricity, massage, and cold baths with friction ("cold rubbing"). For the epileptic attacks large doses of bromid of potassium are decidedly beneficial. This may be combined with chloral, belladonna, etc.

2. **Acute Hemorrhagic Encephalitis in Adults.**—Cases of acute hemorrhagic encephalitis certainly occur in adults. They have been observed either as a sequel of other acute diseases (especially influenza) or as a primary acute disease. In such cases very severe cerebral symptoms (headache, loss of consciousness, hemiplegia) come on quite suddenly and lead to a speedy death with high fever. The autopsy shows several hemorrhagic-encephalitic foci, usually confined to one hemisphere. The brain substance is yellow and softened, very œdematous, and studded with numberless little capillary hemorrhages.

It is of practical importance that, according to more recent observations (the author, Oppenheim, and others), a curable form of encephalitis also occurs in adults. At least, the following class of cases can hardly be interpreted in any other way: Acute onset with fever and grave general cerebral symptoms (headache, stupor, delirium, occasionally deep sopor), also marked focal cerebral symptoms (hemiplegic disturbances, aphasic conditions, hemianopsia, symptoms of cortical irritation, etc.), and, after some time (a few days to several weeks), an entire disappearance of all symptoms with the exception, perhaps, of certain residual symptoms, which, however, do not progress. An ophthalmoscopic examination is of great diagnostic importance in all these cases. It frequently, though not invariably, shows a marked optic neuritis with more or less marked venous congestion.

Naturally, a favorable prognosis cannot always be made with certainty in these cases, and occasionally, even after apparent improvement, death may occur unexpectedly and suddenly. The treatment can be only symptomatic (ice bag, eventually local depletion, antipyrin and similar remedies, narcotics, later on electricity, baths, etc.).

3. **Acute Superior Hemorrhagic Polioencephalitis.**—Wernicke and others have described a form of encephalitis under this name which can be fairly sharply defined clinically, and which is principally observed in alcoholic subjects (whisky drinkers). But other toxæmias (particularly sausage, fish, and

similar poisons) occasionally may produce a similar clinical picture. This form of encephalitis has also been observed as a sequel to influenza and other infectious diseases. The symptoms consist of general cerebral manifestations (delirium, somnolence, headache), combined with paralysis of the ocular muscles of acute onset. At times symptoms of irritation (nystagmus) also appear in the ocular muscles. In some cases an almost complete bilateral ophthalmoplegia develops. The internal ocular muscles (sphincter iridis) and the levator palpebræ superioris are generally spared. At times nervous disturbances, which in many ways suggest the previously discussed alcoholic polyneuritis (*vide* page 273), appear simultaneously in the extremities. The termination of the disease is generally unfavorable. Death may ensue as early as from one to two weeks after the onset of the disease, with symptoms of respiratory paralysis or cardiac weakness. The autopsy shows a hemorrhagic encephalitis chiefly in the vicinity of the third ventricle and the aqueduct of Sylvius—i. e., in the area of the nuclei of the ocular muscles. A termination in recovery may occur, but is rare.

4. **Diffuse Cerebral Sclerosis.**—Diffuse sclerosis of the brain is a peculiar disease, which is usually classed as a chronic inflammation. The whole brain may be involved, or the disease may be confined chiefly to a large part of one hemisphere. There is a very marked increase in the consistency of the brain substance, so that it cuts like a tough piece of leather. The microscope reveals in many cases, but not in all, diffuse hyperplasia of the neuroglia. In a case which we recently examined there was a decided atrophy of the nervous fibers lying in the white substance of the brain. The disease is rare, and its characteristic symptoms cannot yet be stated absolutely. They are developed slowly. The most constant among them seem to be hemiplegia or paraplegia without much change in sensation; symptoms of motor irritation, such as epileptiform convulsions (sometimes general and sometimes unilateral), tremor, or single twitchings, which may be rhythmical or like those of chorea; and, lastly, dementia. When both hemispheres are involved, there are usually marked spastic symptoms in the lower limbs.

The disease has been observed especially in children and also in elderly people. Alcoholism and hereditary syphilis seem to be among the causes, but nothing definite is known upon this point. Transitional forms between diffuse sclerosis and pseudo-sclerosis apparently occur (*vide* page 273). Treatment can be only symptomatic.

CHAPTER VI

INSOLATION. SUNSTROKE. HEAT PROSTRATION. THERMIC FEVER

[**Ætiology and Pathology.**—Under exposure to undue heat, either in the direct rays of the sun, or, during hot weather, in engine rooms, laundries, and the like, marked effects may be produced on the human organism, manifesting themselves in a widely different manner in different cases. The liability to these effects is much greater with us than in most portions of Europe or in Great Britain, and is enhanced by a moist atmosphere which tends to prevent evaporation from the surface of the body. Attacks may come on at night and under cover as well as by day. While excessive heat is the sole and sufficient exciting cause of the changes and symptoms to be described, exhaustion due to muscular exertion or other cause plays a very important secondary rôle. A vigorous person, however, of regular and temperate habits, can stand much greater heat and exertion than an individual who is debilitated or addicted to the use of stimulants. The frequency of attacks after a full meal has been noted. Those newly arrived in the country are, other things being equal, more likely to succumb than natives or those who have become accustomed to the climate. That high temperature due to solar or artificial heat, or a combination of the two, is the prime causative condition, has been clearly shown by experiments on animals. There is practically nothing to add to the researches of H. C. Wood.

Pathological Anatomy.—In cases of sudden death from shock there are no constant and peculiar lesions. The blood is dark, imperfectly coagulated, and collected in the veins; ecchymoses are frequently found.

After death, due chiefly to abnormally high temperature—that is to say, in cases of thermic fever—the heart, and especially the left ventricle, is firmly contracted from post-mortem coagulation of its myosin; the lungs are apt to be much engorged with dark fluid blood, and they may be the seat of hemorrhage; extravasation of blood under the skin is more or less marked, and hemorrhage into and about the cervical sympathetic ganglia has also been observed. The membranes of the brain and cord are often greatly congested, and there may be evidences of beginning meningitis. The blood corpuscles are crenated and show a diminished tendency to the formation of *rouleaux*. Rigor mortis is marked and early in the voluntary muscles as well as in the heart, and is attributable to the same cause. Parenchymatous degeneration of the organs is sometimes found.

Symptoms and Course.—The onset of the attack is usually sudden, though there may be slight premonitions, such as dizziness, pain, or uncomfortable sensations in the head.

It will, perhaps, add to clearness to distinguish three leading forms of attack, it being understood that nature does not always classify cases as sharply as is done here. The first of these comprises cases of heat prostration, denoted by faintness, syncope, nausea, and sometimes vomiting, with marked muscular and general weakness. The surface of the body is cool, the pulse rapid and feeble. The great majority of these cases are mild, and the symptoms pass away more or less quickly on placing the patient in the recumbent posture in a relatively cool and quiet place with free ventilation. After a few hours the patient can be removed to his home, and in a day or two he has recovered perfectly, except for some sensitiveness to heat or the sun's rays. There may be transient insensibility, or, on the other hand, the case may terminate fatally very soon from general exhaustion and cardiac failure.

The second form includes cases with respiratory as well as circulatory failure, due especially to the exhaustion of the nerve centers presiding over these functions. Under this head comes cases of true sunstroke—patients suddenly losing consciousness while exposed to the sun. The skin is cold and the pulse is feeble; death may quickly ensue or recovery may follow, especially if prompt and suitable treatment be instituted. The after-effects of the attack may be very slow to pass away, and may never disappear entirely.

The most striking characteristic of the third form is the great increase in the temperature—a symptom which has given rise to the term thermic fever. In this form premonitions are more common than in the others. The thermometer may register 110° F. or even more; the skin is burning hot and generally dry; the pulse is slow and full, or quick and jerking; the respiration is quickened, sighing, or even stertorous; the pupils at first are usually contracted; there may be great restlessness; coma and convulsions sometimes occur; vomiting is common; and, toward the close of life, the sphincters are sometimes relaxed. A fatal result is due to apnœa and asthenia combined. What was said with regard to recovery from the second form holds good also for the third.

Diagnosis.—This is seldom difficult. The circumstances under which the attack comes on are generally patent; and the hyperpyrexia, if present, is distinctive. Acute alcoholism and meningitis are the chief affections which might lead to error.

Prognosis.—The mortality rate of the severer forms of the disease is very high, but the prognosis depends much on the possibilities of securing prompt and skillful treatment. Under this many a case recovers from a seemingly desperate condition. The tediousness and imperfection of recovery have been already alluded to. For long periods in some cases the mental and physical powers are much impaired, and great care has to be exercised as regards exertion, high temperature, and the sun's rays. Insanity is sometimes a sequel.

Treatment.—In the first place, much may be done by preventive measures to obviate the necessity for any treatment. A regular and temperate life will do much, and special precautions of an obvious nature should be taken during hot weather by those whose occupations involve a liability to exhaustion and exposure to unusual heat. It would be well if it were generally known that there is comparatively little danger of sunstroke so long as perspiration is free. Many an attack might be averted by noting the activity of the skin and seeking rest and shelter as sweating diminishes. A considerable responsibility rests upon militia surgeons and others in similar positions during hot weather.

Mild cases of heat prostration seldom require other measures than those already indicated in describing the symptoms. If there be nausea, and it seem desirable to give stimulants, they are better given under the skin or by the rectum. The clothing should be loosened; the cold douche and other refrigerating measures are not indicated unless there is fever, and they should then be used with caution.

For true sunstroke treatment should be active and energetic, the indications being to reduce the temperature of the overheated centers and to stimulate their activity. If a cool or shady spot be near at hand, the patient should be removed to it and largely stripped of clothing; if not, no time should be lost before resorting to the cold douche on the head and body while stimulants are administered by enema or subcutaneously. External stimulation by mustard or flagellation, and purgative enemata, are said to be sometimes useful. The use of cold externally should not be prolonged in these cases unless there is fever. Nervous exhaustion is the prominent symptom, and all depressing measures are out of place.

The case is widely different with the third form of the attack. Here the immediate danger is from the hyperpyrexia, which must be combated by rubbing the patient with ice, placing him in a tub of water with lumps of ice, or similar measures, until the temperature in the rectum is reduced nearly, but not quite, to the normal point. In the application of the refrigerating measures the head must not be neglected. The sole indication at first is the reduction of the temperature. Antipyrin subcutaneously has been given in a few cases in the Boston City Hospital, and also in Brooklyn, with good results. After a reduction of the temperature, any symptoms of collapse or exhaustion demand stimulants.

With regard to the employment of bloodletting there is considerable difference of opinion. That cases occur in which this procedure is indicated is undoubtedly true, but they are exceptional; they are characterized by the evidences of great cerebral congestion without hyperpyrexia.

The subsequent management of convalescents from any form of sunstroke is often very important. Prolonged rest, and sometimes change of climate, may be demanded. A symptomatic and common-sense treatment is in place. It has seemed to me that quinin, especially in solution with a moderate excess of sulphuric acid, is distinctly useful in those suffering from mild or moderate after-effects of undue heat.—S.]

CHAPTER VII

TUMORS OF THE BRAIN

Ætiology.—The precise causes which lead to the development of tumors in the brain are no more definitely known than in regard to other organs. The new growths are usually formed insidiously and gradually, without ascertainable cause, in persons previously healthy. We are therefore disposed at the present time to assume that there is some abnormal embryonic predisposition of the tissues which acts as the special cause of the subsequent new growth. A circumstance which deserves mention is that sometimes the first symptoms come on immediately or a short time after some injury to the head. Especially in cases of sarcoma arising from the periosteum, the meninges, etc., is a traumatic origin (that is, the "setting free" of a predisposition to tumor by the injury) very probable, although, of course, hardly ever quite certain. Glioma is also referred at times to a traumatic cause.

Most cerebral tumors are found in persons in youth or middle life. Certain forms of tumor, however, particularly solitary tubercles, are comparatively frequent in children. Sex seems to exert a decided influence: men are much oftener affected than women.

Varieties of Cerebral Tumor.[1]—The most important forms of tumor seen in the brain are as follows:

1. GLIOMA.—Glioma is a kind of tumor peculiar to the central nervous system, but seen much oftener in the brain than in the spinal cord (*vide* page 414). The new growth always originates in the neuroglia, which is the con-

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[1] From a clinical standpoint, the term "cerebral tumor" usually includes also such tumors as originate in the neighborhood of the brain (as in the base of the skull), and finally involve the brain itself.

nective-tissue framework of the nervous matter. As seen under the microscope, a glioma is made up of fibers and cells, the latter being like the normal cells of the neuroglia, while the fibers seem to consist mainly of the numerous cell processes. Whether the ganglion cells also take an active part in the new growth has not been definitely proved. It is characteristic of gliomata that they are seldom sharply defined, but shade off gradually into the healthy tissue. The affected portion of the brain is usually enlarged, but yet maintains pretty nearly its original shape. On cross section, the parts that have undergone "gliomatous degeneration" present a yellowish or reddish-gray surface. The growth is usually rather soft, and almost always is very vascular. This great vascularity is not without clinical importance, for variations in the fullness of the blood vessels, and, above all, sudden hemorrhages into the anterior of the new growth, which not infrequently occur, may produce marked symptoms.

Gliomata are most frequent in the white substance of the cerebral hemispheres, but they are also found in the central ganglia, cerebellum, and elsewhere. They are usually single, seldom multiple.

2. SARCOMA.—It is very seldom that any form of sarcoma originates in the brain substance. It usually begins in the connective tissue of neighboring parts, in the dura mater, the periosteum of the cranium, or the cranium itself (osteosarcoma). The sarcoma is found most frequently at the base of the skull, in the form of a circumscribed tumor of varying consistency, and by pressing upon neighboring parts, or by involving them in the diseased process, it may cause the gravest disturbances. Histologically, we have here such varieties as round-celled or spindle-celled sarcoma, fibrosarcoma, etc.

3. GUMMA AND SOLITARY TUBERCLE.—Both gumma and solitary tubercle are very prone to attack the brain. We shall revert to gumma in the chapter on cerebral syphilis. The tubercles may grow to the size of a cherry or larger. They may be single (solitary) or multiple, and may occupy any part of the brain. They are found most frequently in the cortex, and in the cerebellum and pons.

Solitary tubercles and gummata, upon cross section, have a yellowish, cheesy appearance, and are usually distinctly defined. Histologically, they are composed of granulation tissue. It was formerly no easy matter to distinguish gumma from tubercle in all cases, but now certainty is attainable by determining the presence of tubercle bacilli in the latter.

4. CARCINOMA.—Carcinoma completes the list of such cerebral tumors as have much clinical importance. It is always secondary. It has been our experience that secondary cancer of the brain is associated principally with primary cancer of the breast, or of the lungs and pleura, which fact bears a remarkable analogy to the occurrence of secondary cerebral abscess after primary empyema, pulmonary gangrene, etc.

5. RARE TUMORS.—Among the rarer varieties of tumor is *psammoma,* which usually starts from the meninges, is hard, generally comparatively small, and therefore often harmless, and contains calcareous matter, so that it grates under the knife. The calcareous deposits are found in the form of roundish objects, which are composed of flat cells which lie over one another like the coats of an onion. Psammoma is probably of endothelial origin. *Cholesteatoma,* which has a sheen like mother-of-pearl, has also been observed in rare instances, and also *angiosarcoma, angioma,* etc. The not very rare

growths arising from the hypophysis cerebri ("struma of the pituitary gland") remind us of adenomata in their structure.

6. Cysts.—In addition to true tumors we may mention cysts of the brain. They develop in very rare cases without known cause (perhaps as a sequel to cranial injuries?); they may grow to the size of an apple, and may cause precisely the same clinical symptoms as a tumor of the brain.

The General Symptoms of Cerebral Tumors.—As is the case with all focal diseases of the brain, some of the symptoms of cerebral tumors are connected with the special localization of the new growth. There are definite focal symptoms, varying with the part destroyed, or at any rate functionally impaired, and it is from these symptoms alone that we are enabled to determine the position of the tumor. But, in addition to these focal symptoms, there are certain general symptoms common to almost all cerebral tumors of any size. They are in large part referable to the general compression of the brain due to the enlargement of the new growth. In the first place, numerous clinical facts indicate that, whenever there is a tumor of any great size, a large part of the entire encephalon is subjected to pressure; and, secondly, the anatomical appearances of almost every brain affected by a large-sized tumor lead to the same conclusion. The convolutions are flattened and obliterated, the dura mater is crowded against the cranium, and perhaps thinned or even perforated because of the persistent pressure, or, on the other hand, thickened as a result of chronic inflammation. Now and then the effects of pressure are visible even in the bones of the skull; they are worn thin, or even perforated, or their sutures are loosened. Another result of the general intracranial tension, through its effect upon the venous trunks of the brain, is serous effusion into the ventricles (internal hydrocephalus), which occurs very frequently. The largest effusions are caused by tumors in the posterior cerebral fossa, which directly compress the venæ Galeni.

The symptoms of cerebral tumors, referable to the effects of general compression, are as follows:

1. *Headache* is one of the earliest and most constant symptoms. It is usually persistent, although subject to temporary exacerbations and intermissions. The patient describes it as dull, deeply seated, and stupefying. Although it affects the whole head, it is sometimes (not invariably) referred mainly to the neighborhood of the tumor. It is particularly true of persistent occipital headache that it indicates a new growth in the posterior fossa. Sometimes, also, it is possible, by tapping carefully upon the skull, to find an especially tender region. Considerable caution, however, should be used in drawing diagnostic conclusions from such observations. The headache usually lasts to the close of the disease, and, even after the patient has become completely comatose, the persistence of the pain is still evident from his low groans and the way in which his hand seeks his head.

2. Next in frequency to headache are *disturbances of the sensorium and intelligence*. Even the facial expression may be somewhat characteristic, being peculiarly languid, apathetic, and dull. The patient talks slowly, often having to think a long time before knowing what to say. Memory becomes impaired, especially with regard to the most recent events. The interest of the patient in those about him, and the things he used to care for, grows less and less. He has a sleepy, dazed look, and grows careless and untidy. There

is sometimes pronounced somnolence. Of course, individual cases present various deviations from this outline, but in general all cases are a good deal alike, although the degree of mental disturbance may vary from the slighter forms of stupor to the highest degrees of mental weakness. We occasionally see other forms of mental disturbance in tumors of the brain, such as melancholy, hallucinatory confusion, states of excitement, abnormal attempts at wit (Witzelsucht), etc. In such cases, however, local disturbances (especially in the frontal lobe) are probably to be taken into account.

If unusual fullness of the blood vessels, a hemorrhage into the new growth, or some similar cause, induces a sudden temporary increase of tension, there may be such marked symptoms as syncope or an apoplectiform shock.

3. Other general cerebral symptoms are *vertigo, slowing of the pulse,* and *vomiting.* If, however, vertigo be a very prominent symptom, it implies that the cerebellum is especially encroached upon by the tumor. The retardation of the pulse is a frequent symptom, and not without diagnostic value. It has already been mentioned as one result of general compression of the brain in connection with cerebral hemorrhage. The rate may be put at about fifty to sixty, or even lower. The pulse is also sometimes slightly irregular. Cerebral vomiting may be one of the earliest and most troublesome symptoms. It frequently occurs independently of the ingestion of food, especially in the morning, and is not infrequently associated with dizziness.

4. *Epileptiform convulsions* are sometimes excited by cerebral tumors, although many patients are free from them. Such attacks in all probability originate invariably in the cortex of the cerebrum, and it is therefore natural that they should be seen most frequently (as they are) in connection with tumors in the cerebral hemispheres. This rule, however, is not without exceptions. If the convulsions are not general, but unilateral or confined to distinct portions of the body, they are to be regarded rather as focal than as general symptoms, and they may be utilized to determine the position of the lesion (*vide* page 475). A certain amount of information in the same direction may also be obtained from those attacks which begin unilaterally or in one particular limb, and then quickly involve the entire body.

5. *Optic neuritis* ("choked disc," *vide* Fig. 209) is one of the most important and common general objective symptoms of cerebral tumor, occurring in about eighty per cent of all cerebral tumors. We should therefore never omit to make an ophthalmoscopic examination of the fundus oculi in a case of chronic cerebral disease. Some differences of opinion yet exist in regard to the special pathology of optic neuritis, but we may regard it as extremely probable that the main factor in its production is the purely mechanical one of general compression of the brain. This opinion is supported especially by the fact that even a marked choked disc may disappear completely in a short time, if a lessening of the intracranial pressure be brought about by trephining the skull and causing an abundant discharge of cerebro-spinal fluid. The original view of von Graefe was that the increased intracranial pressure obstructs the venous return through the vena centralis retinæ into the cavernous sinus. The opinion which prevails at present is that advanced by Schmidt and Manz—namely, that the increased tension forces the cerebro-spinal fluid into the lymph sheath of the optic nerve (Schwalbe), and that the "*hydrops vaginæ nervi optici*" thus produced compresses the nerve and the blood vessels

which traverse it. At any rate, optic neuritis is not a focal symptom. The tumor occasioning it may have any position if only it gives rise to general compression of the brain.

Disturbances of vision may be entailed by optic neuritis, but they are not a necessary consequence of neuritis; the patient may have amblyopia, defects in the field of vision, or even total blindness. [Cushing has found an interlacing of the color fields is of frequent occurrence in cases of cerebral tumor, before there is any visible neuritis. The boundary of the field for red, for example, may in certain parts of the field be outside the boundary of the field for blue, and in other parts of the field be within it. The boundaries of the color fields usually show some contraction as well.—K.] Only in a few instances has amblyopia been one of the earliest symptoms of cerebral tumor, so that the patient has applied first of all to an oculist. Usually the sight is preserved for quite a long time in spite of the abundant objective evidences of optic neuritis. The latter consists of swelling of the disc, marked distention and tortuosity of the veins, possibly hemorrhages (from passive congestion), and cloudiness of the disc, although the retina still exhibits its normal transparency. It is not until the long-continued congestion impairs nutrition to such an extent as to cause atrophy of the optic nerve that vision is much impaired.

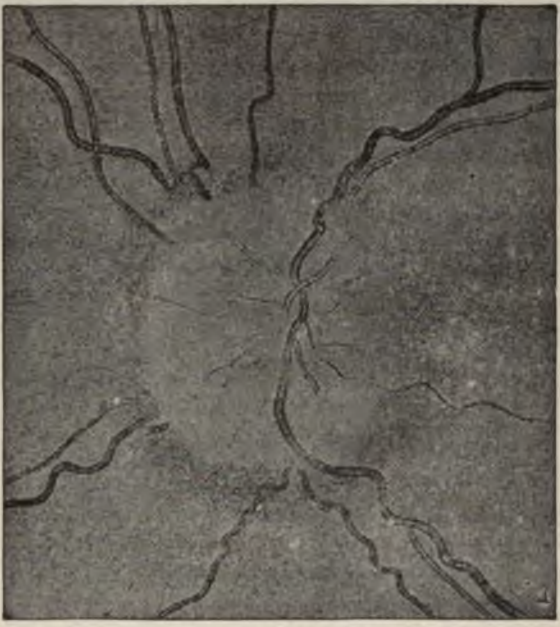

FIG. 209.—Optic neuritis (choked disc) in tumor of the brain (after GOWERS).

6. General *loss of flesh and strength* may sometimes appear comparatively early in cases of tumor of the brain. It is due in large part to the small amount of food taken, vomiting, and wakefulness; but it is not impossible that the grave cerebral disorder itself exerts a direct and unfavorable influence upon all the processes of nutrition. There is in most cases, also, a tendency to obstinate constipation.

7. In conclusion we may call attention to the fact that, on examination of the *spinal cord* of persons who have died of cerebral tumor, degenerative changes have been found in the posterior columns and posterior roots (Dinkler, Mayer, and many others), besides the ordinary secondary descending degenerations. These changes are probably a consequence of the increased cerebral pressure and the stasis of cerebro-spinal fluid thus occasioned, and thus they may be classed among the "general symptoms" of tumor. As a rule they

have no great clinical significance. Only the loss of the patellar reflex, which has been observed in some cases of brain tumor (particularly in tumors of the cerebellum), probably depends on the secondary degeneration of the posterior roots and posterior columns.

Tumors in the Different Parts of the Brain and their Focal Symptoms.—The symptoms above discussed indicate the existence of a tumor, but not its particular location. Other phenomena are necessary to enable us to localize the disease, but it is not exceptional to have none but the general symptoms. Tumors in the white matter of the frontal lobe, or tumors of the corpus striatum or optic thalamus, as well as others, may run their course without any focal symptoms; but most cases afford evidence which points with more or less certainty to the exact position of the tumor. Almost all of these focal symptoms have already been fully discussed in Chapter II of this section, and their interpretation follows the rules for all focal lesions of the brain. We may therefore be brief here. It is necessary only to emphasize the fact that with regard to cerebral tumors, as well as other lesions, focal symptoms should be divided into the direct and the indirect. Direct focal symptoms are the immediate result of the destruction of nervous tissue by the new growth, while the indirect symptoms are excited by the pressure exerted by the tumor upon the parts closely surrounding it. This pressure varies with the amount of blood in the vessels of the new growth, and therefore the indirect symptoms may undergo temporary exacerbations and remissions. An intermediate position is occupied by those focal symptoms which occur in many cases as the result of certain anatomical changes secondary to the new growth. Not infrequently there is white softening of the substance of the brain around the tumor proper. Probably this condition is generally the result of a compression of the minute blood vessels surrounding the new growth, but sometimes it is the sequel of an obliterative endarteritis (Friedländer). The latter cause is especially operative when the tumor is a gumma or a solitary tubercle. In vascular new growths also, especially in gliomata, hemorrhages may occur which may often exert a destructive influence over a greater territory than the new growth itself.

1. Tumors of the Cerebral Hemispheres.—Tumors in the vicinity of the *central motor convolutions* are especially important, because they give the best opportunity for an accurate topical diagnosis and surgical treatment. The first focal symptoms are usually attacks of partial epilepsy, such as have already been described in detail (page 475). Monoplegia gradually sets in, which may slowly develop to complete hemiplegia. At first there is, for example, a brachio-facial or a crural monoplegia, and later the paralysis attacks the leg or the arm on the same side. The precise peculiarities of cortical spasms and paralyses have been described above (see page 475). On careful examination, disturbances of the tactile sense, and particularly of the muscular sense (sensations of motion and position) as well as of the so-called stereognostic sense, can often be discovered in the paralyzed limbs. Ataxia and, with tumors of the left hemisphere, disturbances of speech may also occur. Tumors which involve the *temporal lobes* cause soul deafness or amnesic (sensory) aphasia. Tumors of the *occipital lobe* may be recognized by the occurrence of disturbances of vision (soul blindness, hemianopsia). Marked disturbances of the sense of smell (loss of the sense of smell or

subjective sensations of smell) point to a lesion of the gyrus fornicatus in cases where a direct lesion of the olfactory nerve cannot be assumed. In tumors of the *frontal lobe* Bruns and others have repeatedly observed a striking disturbance of the gait (a staggering gait, a tendency to fall to one side or backward, similar to cerebellar ataxia). This is probably connected with a disturbance of the motor centers for the muscles of the trunk, which are situated in the frontal lobe. Whether certain psychical disturbances (simple dementia, the so-called attempts at wit (Witzelsucht)) occur with particular frequency in frontal tumors is still doubtful. Tumors of the brain not very infrequently arise in the *corpus callosum,* but these can hardly ever be diagnosticated with certainty. Characteristic focal symptoms are lacking; there are usually only the general symptoms of a tumor and especially profound disturbances of consciousness (stupor, somnolence, loss of intelligence) [and paresis, affecting first one side and then the other, with the absence of any involvement of the cranial nerves, etc.—K.]

2. TUMORS AT THE BASE OF THE BRAIN.—The base of the brain is a favorite seat of new growths. The symptoms are in a majority of cases quite characteristic. Some of the tumors spring from the base of the skull; among these are many sarcomata and syphilitic growths ("gummatous periostitis"). Other tumors originate in the meninges, especially the dura; and still others from the brain itself. Of these last, it is remarkable that some spring from the pituitary gland. The exact starting point is very seldom of much clinical importance, for all the parts mentioned are in such close proximity to one another that there is no great difference in the symptoms produced. We can decide merely that there is a tumor in this or that place at the base of the brain.

Tumors at the base of the brain owe their characteristic clinical stamp to the frequency with which the cranial nerves at the base are involved. The anatomical relations are such that these nervous trunks are often compressed by the new growth or actually incorporated in it. Of the symptoms thus occasioned, the most frequent is paralysis of the motores oculi (oculomotor and abducens). This is at first usually unilateral, but it may later affect both sides. If one of the optic tracts be involved, hemianopsia may result, and pressure upon one optic nerve may produce unilateral choked disk with unilateral disturbance of vision. If both optic tracts are affected, peculiar limitations of both visual fields result, which can generally be easily explained by the well-known course of the optic fibers in the optic chiasm (*vide* page 477). Lesions of the trigeminus not infrequently produce sensory disturbances in the face, and in isolated cases also paralysis of the muscles of mastication. Fibromata occur which arise from the sheath of the trigeminal nerve, very much like the acoustic tumors which will be described shortly. The trunk of the facial often suffers. The facial paralysis thus occasioned throws considerable light upon the diagnosis, for the frontal muscle and the orbicularis oculi are involved (compare page 504), and there is usually to be found in the paralyzed muscles the reaction of degeneration, showing that the paralysis is peripheral. We have therefore reason to assume that the lesion is situated at the base of the cranium rather than in the brain itself. Tumors arising from the posterior fossa at the base of the brain cause pressure paralysis of the lower cranial nerves (hypoglossus, accessory) and sometimes pronounced bulbar symptoms (disturbances of swallowing and articulation) from pressure on the medulla. As might naturally be expected, various degrees and forms of paralysis in the extremities are often found in combination with all the above-mentioned disturbances of the cranial nerves. Such conditions are most frequent when the crus cerebri, with its pyramidal tract, is affected. There is no need of enumerating all the possible varieties of symptoms. We must consider them all carefully in each individual case, and then, by bearing in mind the anatomy of the parts, we shall, in a majority of instances, be enabled to determine with some approach to accuracy the place at the base of the brain where the new growth must be. Sometimes we may be led into error by tumors which, though situated in the brain substance and at a comparative distance, yet by their pressure give rise to indirect symptoms referable to the cranial nerves at the base.

The tumors of the hypophysis are of particular interest on account of their relation to acromegaly (*q. v.*). But tumors of the hypophysis without concomitant acromegaly are also by no means rare. The most characteristic symptoms are peculiar disturbances of vision in consequence of the pressure of the tumor on the chiasm. Bitemporal hemianopsia from pressure on the posterior angle of the chiasm, or complete unilateral blindness with hemianopsia of the other eye, are disturbances which so far have been observed almost solely in tumors of the hypophysis. Atrophy of the optic nerve can generally soon be demonstrated with the ophthalmoscope. Psychical changes (confusion, somnolence) are the most prominent of the general tumor symptoms. In cases without true acromegaly, nutritive disorders should be looked for, viz., glycosuria, adiposity, myxœdematous changes, loss of hair, subnormal temperature, etc. Disturbances of the sexual sphere (amenorrhea, anomalies of the genitals and breasts) are particularly interesting. Sexual anomalies are also observed in tumors of the pineal gland. The deepening of the sella turcica of the skull in hypophysis tumors has been repeatedly demonstrated by means of the Röntgen rays.

3. TUMORS IN THE NEIGHBORHOOD OF THE THIRD VENTRICLE.—Tumors sometimes develop which are situated in a central position in the interior of the brain, especially in the neighborhood of the third ventricle, and therefore give rise to few well-marked focal symptoms. Besides the general symptoms of tumor (amaurosis from optic neuritis, stupor, headache, vomiting) there is general weakness of the extremities, often associated with spastic rigidity, which is usually only a little more developed on one side than on the other. As a rule, therefore, it is hard to make an accurate topical diagnosis. Tumors of the corpora quadrigemina cause bilateral paralysis of the ocular muscles, particularly often of the same muscles on both sides; in addition, disturbances of vision and hearing, pupillary changes and ataxia of the trunk on walking and standing.

4. TUMORS OF THE CEREBELLUM.—Cerebellar tumors are comparatively frequent, and they can usually be diagnosticated with considerable certainty. The general symptoms of tumor (headache, vomiting) are, as a rule, very pronounced. The headache is intense and continuous, is generally located in the occiput and nape of the neck, and is not infrequently associated with marked cervical rigidity. The skull is often sensitive to percussion over the occiput, and the difference in tenderness of the two sides (sometimes also

differences in the percussion note) may be used with caution in determining the seat of the lesion. A marked choked disc, which generally appears early, and is often associated with disturbance of vision, is also characteristic of cerebellar tumors. In cases of bilateral disturbances of smell and diminution of hearing, which have been observed several times in tumors of the posterior fossa, we may possibly think of analogous symptoms due to congestion of the acoustic or olfactory nerves. But these nerves may also be compressed by the associated secondary internal hydrocephalus, which is generally very marked. The general pressure symptoms of cerebellar tumors, particularly the headache, vertigo, and vomiting, often depend evidently on the particular position of the body and the head. The patients consequently frequently assume a definite position of the body and the head, in which, according to their experience, they have the least disagreeable symptoms. Vertigo and cerebellar ataxia of the trunk are the most important cerebellar focal symptoms. Their significance has been already explained in detail (see page 492). Cerebellar tumors not infrequently, as they continue to grow, exert pressure upon the pons and medulla, and thus special clinical symptoms may arise: symptoms of paralysis or irritation on the part of the posterior cranial nerves, nystagmus, paralysis of conjugate associated movements of the eyes, hemiparesis, hemiataxia, etc.

In connection with cerebellar tumors the tumors of the cerebello-pontine angle deserve special mention, especially since they have repeatedly been correctly diagnosticated and successfully removed by operation. They are generally benign fibromata or fibrosarcomata, which start from the sheath of the acoustic nerve. The symptoms of the disease therefore begin, as a rule, with disturbances of hearing (unilateral nervous deafness, tinnitus) or vestibular symptoms (vertigo, unsteady gait), to which later on are added symptoms in the facial nerve, the trigeminus (particularly often a unilateral disappearance of the corneal reflex), the abducens, and, furthermore, in the cerebellum and pons (nystagmus, paralysis of conjugate associated movements of the eyes). Disturbances of deglutition and articulation may arise from pressure on the medulla. The general symptoms of tumor (headache, choked disc) are in many cases insignificant for a long time.

5. Tumors in the Pons, the Crura Cerebri, and the Medulla.—These tumors are to be judged according to the general rules for the localization of cerebral diseases. Tumors of the pons are chiefly gliomata or solitary tubercles. The general symptoms of tumor are, as a rule, not very pronounced. Choked disc, especially, is generally absent. The focal symptoms which have the greatest diagnostic significance, are crossed hemiplegia (hemiplegia alternans)—i. e., paralysis of the extremities of one side, paralysis of one facial, abducens, or trigeminus of the other side, and paralysis of the conjugate associated movements of the eyes (lesion of the abducens nucleus). If the fibers of the lemniscus are affected, sensory disturbances and hemiataxia result.

We may also observe, by way of an appendix, that repeated attempts have been made of late to make use of percussion of the skull as an aid in the diagnosis of the seat of a cerebral tumor. Over a tumor which is not too deeply seated the bones of the skull are said to give sometimes on percussion a note which is not wholly dull but rather tympanitic. A sort of "cracked-pot" resonance, due to a separation of the sutures, has also been observed. Percussion of the skull, however, has no great practical significance, since the interpretation of the results is too uncertain.

[In view of the importance of the focal diagnosis of tumors of the brain, it has seemed advisable to insert the following brief summary of the focal symptoms of tumors in different parts of the brain:

Prefrontal Region.—Marked mental impairment (abnormal attempts at wit [Witzelsucht] ?); symptoms of invasion of the central region (Jacksonian epilepsy, aphasia); ataxia; disturbances of smell.

Central Region.—Jacksonian epilepsy; monoplegia or hemiplegia; tactile hypæsthesia and loss of stereognostic sense; motor aphasia.

Posterior Parietal Region.—Word blindness; disturbance of muscular sense(?); homonymous hemianopsia.

Occipital Region.—Homonymous hemianopsia; soul blindness.

Temporo-sphenoidal Region.—Often a latent region. Word deafness, if the left side be affected; disturbances of taste, smell, and hearing.

Corpus Callosum.—Often a latent region. Progressive hemiplegia, often bilateral from invasion; mental disturbances.

Optico-striate Region.—Hemiplegia; contracture. In posterior part, hemianæsthesia, homonymous hemianopsia, posthemiplegic chorea, athetosis. In thalamus, hemianæsthesia, especially for muscular sense; hemiparesis; hemiataxia; pain on affected side; choreic movements.

Crus Cerebri.—Crossed paralyses of the oculomotor nerve and limbs.

Corpora Quadrigemina.—Oculomotor paralyses; reeling gait; blindness (?); deafness (?).

Pons and Medulla.—Crossed paralyses of face and limbs, or tongue and limbs. Other cranial nerve lesions.

Cerebellum.—Marked cerebellar ataxia; marked vertigo and vomiting; asynergy; adiadocokinesis.

Cerebello-pontine Angle.—Deafness, tinnitus, vertigo, facial paralysis, trigeminal anæsthesia, abducens paralysis.

Base, Anterior Fossa.—Mental impairment; disturbances of smell and vision; exophthalmus.

Base, Middle Fossa.—Disturbance of vision; oculomotor disturbances; hemiplegia.

Base, Posterior Fossa.—Trigeminal neuralgia; neuro-paralytic ophthalmia; paralyses of face and tongue; disturbance of hearing; crossed paralyses.

Hypophysis.—Disturbance of vision, especially bitemporal hemianopsia; oculomotor disturbances; disturbances of nutrition and of the sexual functions; acromegaly.

It also is of importance, in cases which may come to operation, to determine whether the growth be cortical or subcortical. Seguin has given certain rules which, however, are not absolute. "In favor of a strictly cortical or epicortical lesion are these symptoms, none of them having specific or independent value: Localized clonic spasm; epileptic attacks beginning by local spasm, followed by paralysis; early appearance of local cranial pain and tenderness; increased local cranial temperature. In favor of subcortical location of a tumor: Local or hemiparesis followed by spasm; predominance of tonic spasm; absence, small degree, or very late appearance of local headache and of tenderness on percussion; normal cranial temperature."—K.]

General Course of Cerebral Tumors.—The symptoms of these growths almost always cover a long period of time. Exceptionally a tumor remains latent till a hemorrhage or some similar event takes place in it, giving rise to sudden and grave symptoms, and possibly to an equally sudden termination. The rule is, however, for the phenomena to develop gradually. According to the location of a new growth, either the general or the focal symptoms may come first into prominence. They generally occur in the order named. First of all is an ill-defined, deeply seated headache, and by degrees all the other general and focal symptoms follow after. The symptoms may vary repeatedly and greatly in severity, a fact due mainly to the varying pressure of the tumor on neighboring parts. Repeated mention has already been made of the sudden exacerbations which may come on, especially in case of the vascular gliomata.

The entire duration of the disease is usually at least some months, and may be one or two years or more. The termination is almost invariably unfavorable. Death may be rather sudden, or it may not come till after a long period of wretchedness. Fortunately, however, the lameness, blindness, and marasmus are frequently made less terrible to the patient because of his mental debility and torpor. Recovery is possible only when the growth is syphilitic. It is indeed possible that solitary tubercles may also end favorably, but the matter is still in doubt.

Diagnosis.—The hope of rendering focal lesions of the brain visible by means of the Röntgen rays have thus far not been realized [except in a very few cases.—K.]. The diagnosis of cerebral tumor depends therefore entirely on the symptoms, and primarily on the gradual onset and continuous slow increase of the general symptoms above detailed—namely, headache, vertigo, vomiting, convulsions, dementia, etc. The most constant of these symptoms is the headache. They all indicate the development of some chronic brain trouble, a tumor being the most probable if there be no definite ætiology to suggest some other process, such as traumatism resulting in abscess, or syphilis. Much stress may also be laid on the optic neuritis, since this, as we have said, is much less often seen in other chronic brain diseases (abscess or softening) than in tumor.

The general symptoms indicate that a tumor of the brain exists; but, in order to learn the position of that tumor, we have to rely mainly upon the focal symptoms. Their gradual development and the way in which one new symptom is slowly added to another, also give further ground for the opinion that some continuously progressive disease exists, and most probably a cerebral tumor. Of diseases with a similar course, abscess is recognized especially by the slighter symptoms of general intracranial pressure, and particularly by the frequent, though not invariable, absence of optic neuritis, frequently by febrile symptoms, and by its ætiology (trauma, purulent otitis). Inflammatory and thrombotic softening, if they come on slowly, usually produce less general disturbance than do tumors; they hardly ever cause optic neuritis, and (unless of syphilitic origin) are much rarer before middle age than tumors of the brain. Sclerosis sometimes simulates cerebral tumor; but here also there is no choked disc; the disease lasts much longer (five or ten years or more), and, inasmuch as the sclerosis is usually multiple, there is frequently too great a complexity of symptoms to warrant the assumption of one solitary lesion. [We must, however, bear in mind that, in about one seventh of the cases, tumors also are multiple.—K.]

Certain rare cases of chronic circumscribed meningitis cannot be differentiated from a tumor. They generally occur at the base, lead to a considerable thickening of the tissues, and they may simulate all the symptoms of a new growth in this region. Occasionally, also, chronic hydrocephalus is confounded with tumor of the brain. We met with a case of dropsy of the fourth ventricle which presented during life a perfect picture of tumor of the cerebellum. Certain forms of chronic or subacute meningo-encephalitis which have been studied but little so far, are above all apt to be confused with tumors of the brain. In these cases grave general cerebral symptoms develop (headache, sopor, vomiting) associated with marked optic neuritis. The diagnosis of brain tumor seems to be positive, and, nevertheless, improvement sets in, or at least, there is no progression of the symptoms. Cases of this sort, several of which I have myself observed, are now occasionally designated as " meningitis serosa "; there is very likely, however, an encephalitic process at the basis. Nonne has described similar cases of " pseudo-tumor," in which all the clinical symptoms point to the presence of a brain tumor, while the autopsy discloses an apparently normal condition of the brain.

As to the nature of a new growth, we cannot go beyond surmises. If the focal symptoms indicate that the tumor is in the substance of the brain itself, our first thought would be of a glioma, because it is by far the commonest sort of growth in that situation; and, as has been stated, certain peculiarities in the course of the disease (especially if new symptoms add themselves abruptly) would make glioma probable. If, on the other hand, the tumor be at the base, it is most apt to be sarcoma, which is the most frequent form of new growths here. Fibromata of the acoustic nerve and tumors of the hypophysis should be particularly borne in mind. In all cases, especially in tumors at the base of the brain, we should bear in mind the possibility of syphilis. The previous history and the entire body of the patient should be closely searched with this point in mind; its therapeutic importance need not be dwelt on.

One special form of tumor deserves a brief mention here—namely, large cerebral tubercles. The growth may be single (solitary), or it may be multiple. It is seen chiefly in childhood, and any chronic cerebral disorder in a child should suggest the possibility of such a growth. It is rendered all the more probable by the coexistence of the signs of tuberculosis elsewhere, as in the lymph glands, lungs, or bones. The symptoms are analogous to those produced by other tumors. Among the most frequent phenomena are headache and convulsions. The latter are often unilateral. There may also be all sorts of focal symptoms, according to the locality of the lesion.

Prognosis.—Except gummata, all tumors of the brain have a very unfavorable prognosis. It is said that in very rare instances tubercular growths have been arrested or cured; but in practice we can hardly ever rely upon any such result. In all other cases recovery, aside from the possibility of surgical treatment, is practically excluded. The time intervening between the appearance of the first symptoms and death varies greatly, as has been said. We should therefore be very cautious in predicting the duration of the illness. It seldom, however, exceeds one or two years, and sudden death without any warning may occur at any time.

[Tubercular growths are occasionally found at autopsies, which have become encapsulated, and have apparently existed for years without causing symptoms. In some cases, especially with tubercular growths in children, the symptoms diminish and the patient may live for years, suffering only from the blindness, etc., caused by the growth. Other cases may present only mild symptoms, and the patient may lead a fairly comfortable life for years.—K.]

Treatment.—Inasmuch as the nature of the tumor cannot be determined with absolute certainty in any instance, antisyphilitic treatment may always be tried; 40 to 75 gr. (gm. 3 to 5) of mercurial ointment should be used by inunction, and 30 to 75 gr. (gm. 2 to 5) [or more] of potassium iodid should be given internally each day. If the new growth be syphilitic, much benefit may be obtained in this way. It must be confessed that the treatment is generally of little avail, because the tumors are not of a syphilitic character; although it may be that iodid of potassium sometimes has a temporary good effect upon other forms. A long-continued course of arsenic has also been recommended, in order to check the growth of the tumor. This remedy should be used especially in those cases where there is a suspicion of sarcoma or solitary tubercle. We may try treatment by the Röntgen rays, but without much hope of benefit.

Of late years many attempts have been made to remove tumors of the brain by surgical measures. In this respect the special favorable conditions of the individual case are chiefly to be considered. If we are able to make an approximately certain diagnosis of a tumor on the surface of the brain, especially in the motor cortex, surgical interference is warranted, and in some cases it has been attended with decided success, either permanently or at least for a time. Since there is no outlook for the patient's recovery without an operation, we should make an attempt at operation in every case where the tumor can apparently be diagnosticated with certainty and localized in the motor region of the cortex. We should, of course, never undertake an operation with too great expectations, since errors in diagnosis and unforeseen complications and incidents only too often arise. Of the remaining brain tumors, those of the cerebellum are particularly amenable to surgical treatment. Tumors of the cerebello-pontine angle especially have been repeatedly removed with the best permanent results. Tumors of the hypophysis also have recently been removed with success in several instances (v. Eiselsberg and others). When there are marked general symptoms of pressure (headache, somnolence, optic neuritis) it may be well worth while, as a practical measure, to advise simple opening of the skull without further interference. In some cases the lessening of intracranial pressure that ensues has a very favorable effect, at least upon these general pressure symptoms of tumor. [Decompression should be performed on every case of progressive optic neuritis, in order to prevent blindness.—K.]

Beyond what has just been indicated, treatment must be symptomatic. The headache is combated with ice bags, narcotics, antipyrin, etc.; the convulsions require bromid of potassium or the inhalation of chloroform; the vomiting is lessened by rest in bed, opium, and bits of ice. The general care and nursing of the patient are very important, so that bedsores and the like may be avoided if possible.

APPENDIX

HYDATIDS OF THE BRAIN

It was stated in Vol. I, on page 630, that the cysticercus cellulosæ, which originates from the tænia solium, may occur in great numbers in the brain. The cysticerci most frequently occupy the pia mater, but generally project downward into the cortex of the brain. The meninges not infrequently exhibit signs of chronic inflammation, and may present hemorrhages, which are not always minute. If there are numerous cysticerci in the neighborhood of the ventricles, a varying degree of internal hydrocephalus usually develops. The individual cysticerci are usually enveloped in a capsule of connective tissue, but they may be entirely devoid of such a covering.

No characteristic clinical sketch of hydatids in the brain can be drawn, because the symptomatology of each case differs according to the number and position of the parasites. Sometimes cysticerci produce absolutely no symptoms, and are discovered incidentally at the autopsy. In other instances they cause a long and tedious illness. Epileptiform convulsions seem to be the most frequent symptom, and these must be due to the position of the cysticerci in the cortex of the brain. They may, like true epileptic attacks, come on only at certain times when the general condition is otherwise good; or permanent general cerebral symptoms may also appear, such as persistent headache, vertigo, and mental disorder. In a case recently under our observation the symptoms consisted solely of paroxysmal headache and uncontrollable vomiting. The diagnosis of suspected tumor was made. The autopsy showed a cysticercus the size of a cherry in the fourth ventricle with secondary hydrocephalus. Other cases have shown merely mental disturbances (confusion), staggering gait, vertigo, etc., so that the disease has been repeatedly confounded with hysteria. Special focal symptoms are only rarely present with cysticerci of the brain. It is a fact of great practical significance that death may not infrequently come on suddenly and unexpectedly in cysticerci of the brain. We have seen several such cases in persons who previously had not complained of feeling at all ill, or who had complained merely of slight headache, etc. In such cases the cysticerci are usually in the third or fourth ventricle, and probably cause sudden pressure upon the medulla.

The diagnosis can never be made with absolute certainty. The presence of cysticerci in the brain may be suspected when the above-mentioned severe symptoms occur in a butcher or other person who is from his calling especially exposed to infection, or who is known to have had or still to have a tapeworm, or in whom cysticerci have been demonstrated in some other part of the body, such as the skin.

We know of no remedy capable of destroying the cysticerci. Treatment, therefore, can be only symptomatic. [In a few cases, chiefly in Australia, the cysticerci have been successfully removed by surgical interference.—K.]

CHAPTER VIII

CEREBRAL SYPHILIS

Ætiology.—The importance of syphilis as an ætiological factor in many chronic diseases of the nervous centers has been repeatedly adverted to in preceding chapters; but, as we have already stated, the influence of syphilis upon the genesis of these diseases is shown in two essentially different ways. In one case there is a probable toxic, so-called metasyphilitic degenerative process of certain tracts of fibers and ganglion cells (tabes, general paralysis); in the other case there is the formation of true tertiary syphilitic ("gummatous") new growths. Only this latter form of syphilitic cerebral disease will be spoken of here.

As a rule, genuine cerebral syphilis develops in the later stages of the whole syphilitic process, but sometimes cerebral symptoms come on by the end of the first year from the date of the initial lesion. Usually the interval is several years, and it may be ten or even twenty years after the beginning of the disease before the first signs of the cerebral trouble develop. We therefore class cerebral syphilis generally among the "tertiary symptoms" of syphilis. [Recent investigations have shown that syphilis quite frequently attacks the brain and spinal cord in the very early stages of the disease, a few weeks after the primary infection.—K.]

Liability to the disease does not seem to be influenced essentially by age or sex, except so far as pertains to the general distribution of syphilis. Even hereditary syphilis has been proved to cause diseases of the nervous system. But it cannot be denied that a predisposition to cerebral syphilitic disease is often engendered by those influences which are apt to promote cerebral disease in general. Just as the position of syphilitic cutaneous lesions is often determined by external irritation at some one place on the skin, so the disease seems more liable to attack a brain which is exposed to certain unfavorable conditions than one which is perfectly normal and vigorous. Such conditions are inherited tendency to nervous diseases, various injurious mental influences, poisons, in a broad sense, and traumatism. It need hardly be said that even a previously sound brain is not immune from the affection.

Pathology.—As far as has yet been ascertained, there are two chief forms assumed by syphilis in the brain: (1) A circumscribed, broad, flat syphilitic new growth like a tumor (gumma, syphiloma), and (2) a disease of the arteries of the brain, which is usually quite extensive. There is no essential difference underlying these two varieties. They may also occur in combination with each other. The disease of the blood vessels is really a syphilitic new growth affecting the walls of the arteries.

Syphilitic new growths are yellowish- or grayish-red, and frequently cheesy in the center. Their most frequent seat is the dura mater or the subarachnoidal space, whence they spread to the brain substance or the neighboring vessel and nerves, but exceptionally they may originate in the substance of the brain itself. Histologically they are made up of granulation tissue of varying degrees of vascularity, presenting yellow spots usually visible to the naked eye. These spots are of firmer consistency than the rest of the growth, and have undergone coagulation necrosis (have become cheesy). In the brain substance itself circumscribed, wedge-like gummata may sometimes develop, which closely resemble solitary tubercles; but the extensive, flat syphilitic new growths arising from the meninges (*meningitis gummosa*) are of much greater clinical importance. These are found most frequently at the base of the brain, especially developing from the region of the chiasma, or, more rarely, on the lateral portions of the brain (fissure of Sylvius) or on the convexity. In these more extensive new formations of tissue we usually find all the different stages of the process together, fresh granulation tissue, cheesy spots, and, finally, the transition into contracting cicatricial connective tissue (scar formation).

Syphilitic disease of the arteries was first fully recognized by Heubner, who has described it accurately. It is usually most pronounced in the arteries of the base of the brain, and especially in the middle cerebral artery and its branches. Even the unaided eye detects a grayish opacity in the arteries. They feel firm and stiff, and on cross section their walls are found to be thickened, either uniformly or in some places more than in others. This causes no inconsiderable narrowing of the lumen, or even its obliteration, particularly if the last gap be closed by the formation of a thrombus. The microscope shows that the new growth originates chiefly in the intima of the vessel, where there is a hyperplasia of the endothelium and a gradual transformation of it into a firm connective tissue. But the adventitia also undergoes a gradual thickening of considerable extent. Syphilitic endarteritis presents no distinctive histological characteristics. Entire certainty that the inflammation is syphilitic can be attained only by discovering other evidences of syphilis, either in the brain or elsewhere, or from the personal history and the previous course of the disease.

The great clinical importance of syphilitic endarteritis is due to its cutting off the normal supply of blood from the regions supplied by the diseased arteries. If the occlusion be complete, cerebral softening is inevitable, as in ordinary embolism and thrombosis of cerebral arteries; and, inasmuch as the middle cerebral artery is particularly liable to the disease, syphilitic softening is most often found in the region supplied by this vessel.

Clinical History.—The variety of the pathological processes and of their location produces a corresponding variety in the symptoms of cerebral syphilis; but certain types, already described in the main by Heubner, may be distinguished, and in many cases these present a picture which is very characteristic of cerebral syphilis.

1. *Basal Gummatous Meningitis. Basal Cerebral Syphilis.*—The development of a flat syphilitic new growth spread over the base of the brain, whose anatomical relations have been briefly described above, causes a form of disease which in many respects agrees with that of tumors of the base of the brain (*vide supra,* page 536), but which also often shows certain characteristic peculiarities. The first symptoms consist of persistent headache, sometimes growing worse at night, vertigo, vomiting, and a certain mental dullness, which rarely, however, reaches such a high degree of stupor as is so often seen in genuine tumors of the brain. States of marked maniacal excitement also occur at times. The patient often has an intense and striking polydipsia and polyuria. To these symptoms are soon added others, which are due to the in-

vasion of the different basal cranial nerves by the morbid process. The optic nerve and the motor nerves of the eye (especially the oculomotor) are almost always among the first to be affected. Disturbances of vision (limitation of the visual field, in some cases hemianopsia, blindness of one or both eyes), changes in the pupils, disturbances of motion of the eyeballs or eyelids, may now become manifest in various ways. Optic neuritis or choked disc may occur, but in general the ophthalmoscopic changes are decidedly less frequent in cerebral syphilis than in genuine tumors of the brain. It is a very characteristic circumstance that all the different symptoms on the part of the cranial nerves (especially the changes in the visual field, as Oppenheim has shown) may show great variations, which are undoubtedly connected with changes in the pressure from cicatricial contraction, etc. Of the other cranial nerves, the facial, acoustic, olfactory, and trigeminus are most readily affected, but we should always make a very complete examination in every way in order to estimate correctly the extent of the process.

The further course of the disease may vary very much. In fresh cases, which are recognized and treated in time, there may be decided improvement, and even complete recovery, or at least an arrest of the morbid process; but, on the other hand, the symptoms may increase. If there is also syphilis of the arteries (*vide infra*), there may be a slowly developing hemiplegia, hemiplegia following apoplexy, epileptic states, bulbar symptoms, etc.; in short, a complicated form of cerebral disease no longer capable of any material change.

2. *Gummatous Meningitis and the Formation of Syphiloma on the Convexity of the Brain and in the Vicinity of the Fissure of Sylvius.*—In this somewhat less frequent localization of the syphilitic new growth prodromata similar to those just enumerated precede the severer symptoms. Then appear violent partial or general epileptiform convulsions. These often come on very suddenly, and they may recur at considerable intervals or in quick succession. There are usually still other symptoms of cortical disturbance, especially paresis of one limb or even of one half the body; very frequently slight disturbances of speech (stumbling over syllables), referable to the cortex, and indications of mental impairment. Optic neuritis, as a rule, is not present. Many of these cases reach a fatal termination comparatively early. The epileptiform convulsions become more and more frequent, and unconsciousness increases to deep coma ending in death; but in these cases prompt and energetic treatment may accomplish a great deal.

3. *Cerebral Syphilis Involving the Cerebral Arteries Chiefly.*—A third common and important variety of cerebral syphilis is characterized chiefly by syphilitic arteritis. Not infrequently there is a prodromal stage, which is sometimes only slight; then, as a result of the occlusion of some vessel, which often occurs quite suddenly, there is a pronounced apoplectic attack, followed in most cases by hemiplegia. The intensity of the initial shock may vary greatly; sometimes there is only a slight dizziness, sometimes there is coma that lasts for days. Sometimes the shock is succeeded by a peculiar condition of mental confusion and dullness, which may persist for weeks. In severe cases death is speedy, and is usually ushered in by a marked rise of temperature. Other patients improve more or less rapidly, especially under proper treatment.

Apoplectic attacks of this sort may recur after temporary improvement has taken place, and may be associated with all sorts of nervous symptoms,

4. *Complicated Cases of Cerebro-spinal Syphilis.—Combination of Gummatous Syphilis and Syphilitic Degeneration of the Nerves* (*Tabes, General Paralysis*).—Besides the three types of cerebral syphilis already described there are also many cases which belong in part to one of the types mentioned, but which, on the other hand, exhibit much more complicated symptoms on account of the far greater extent of the anatomical changes. In the first place we will mention the combination of cerebral and spinal symptoms. The latter are often due to gummatous spinal meningitis also, which is very often situated in the cervical region of the cord, and which may lead to paraplegia, brachial paresis, symptoms of unilateral lesion (*vide* page 430), shooting root pains, vesical disturbances, etc. With these symptoms, all of whose varying details it is impossible to speak of here, are not infrequently associated cerebral symptoms, which, for their part, correspond to the conditions above described. A considerable number of cases which have been studied anatomically of late are also of especial interest. In these cases the symptoms of gummatous syphilis were associated with genuine tabes, or in other cases cerebral symptoms ensued, which were of the type of general paralysis (weakness of memory, a peculiar disturbance of speech, twitching of the facial muscles, paralytic attacks, etc.). In such cases the multiplicity of possible details is inexhaustible; but the characteristic peculiarity of the type lies in this regular irregularity, which often admits a positive recognition of syphilis of the nervous system and is also capable of a clinical and anatomical interpretation, with proper consideration of the nature of the disease.

Diagnosis.—The most important diagnostic criteria have already been mentioned in the description of the symptoms. Here, too, the diagnosis is first topical and then ætiological and anatomical. The distinction between syphilis and genuine tumors is the most difficult. Absence of optic neuritis, a marked variability in the severity of the symptoms, and multiplicity of symptoms, especially the combination of symptoms demanding different localizations, are in favor of syphilis. The discovery of a previous syphilitic infection is, of course, always important. We cannot here describe in detail the methods of determining this fact. The history of the patient, as well as previous specific affections—such as, in women, miscarriages, abortions, etc.—and the objective signs on other parts of the body, are the two sources of information. There may be scars on the skin or mucous membranes, enlarged glands, ulcers, tibial periostitis, or changes in the testicles. Age is also important; thus, an apoplectic attack in a young person would suggest syphilis, because the other causes of such an attack operate chiefly upon the aged. The results of treatment often throw considerable light upon the diagnosis. As there is nothing to lose and much perhaps to gain, we should not hesitate to give specific remedies (*vide infra*) in doubtful cases. If they prove successful, the diagnosis of syphilis receives strong confirmation. We can only briefly call attention here to the great importance of the serum diagnosis of syphilis.

Prognosis and Treatment.—There are few severe and dangerous diseases in which timely and appropriate treatment is attended with so much success as that achieved in many cases of cerebral syphilis. In order, however, both to understand the favorable results and not to be misled by the failures, we need to gain a clear idea of the way in which antisyphilitic remedies can be of benefit. They can accomplish this only by causing the dissipation and absorption of the new growth—that is, the gumma or the swelling of the intima. If this be effected, the surrounding parts are, of course, relieved from pressure, and the circulation becomes unimpeded. If the tissues still retain functional power, they resume their functions and all symptoms of disease vanish. But when the tissues have already been considerably impaired by the compression, or by the scanty blood supply, the results are quite different. Even then the nerve trunks at the base of the brain may gradually become regenerated, but such parts of the true cerebral parenchyma as have undergone softening have lost their functional capacity forever. In such cases antisyphilitic treatment is unavailing.

It is therefore obvious that the first essential of success is to begin treatment as early as possible. The sooner a correct diagnosis is reached, the sooner will existing symptoms be relieved, and further danger be averted. The method of treatment which will probably accomplish all that can be accomplished, and in the shortest possible time, is energetic mercurial inunction. At least a drachm (gm. 3 to 5) of mercurial ointment must be rubbed in every day at first, according to the ordinary method. We should not venture to restrict the diet unless the patient be well nourished and "full blooded." If he be anæmic and feeble, a generous regimen is demanded. Usually the internal administration of iodid of potassium is combined with the inunctions; we should give 30 to 45 gr. (gm. 2 to 3), or, in severer cases, even 1 or 1.5 drachms (gm. 4 to 6) daily. [In this country iodid of potassium is given much more freely. In cases where syphilis is suspected, we may safely begin with doses of 30 gr. (gm. 2) three times a day. If the symptoms be urgent, the dose should be rapidly increased until the patient takes 3 or 4 drachms (gm. 10 to 15), or even more, three times a day. If the patient have syphilis, the danger of iodism seems less. The drug must be given in large amounts of water or milk, and it is often better borne if given in Vichy or Giesshübler water. Sometimes the stomach will not tolerate the extreme doses.—K.] The same remedy should also be given afterwards, for a long time, in smaller doses. When there is no benefit at all after twenty or thirty inunctions, there is little prospect of any appreciable improvement. In favorable cases the mercury often begins to produce some effect after the fifth or sixth inunction, and it may cause astonishingly rapid improvement.

Some authors prefer intramuscular injections of mercury (calomel, mercury salicylate), and maintain that they have secured good results in cases in which mercurial ointment failed. Potassium iodid alone is sufficient for the milder cases only, where there is merely headache, trigeminal neuralgia, or paralysis confined to the ocular muscles. Frequently the milder sodium iodid is to be preferred to the potassium iodid. Iodipin, sajodin, etc., are also occasionally used with good results. In many cases it may be desirable for other reasons to carry out the treatment at some health resort rather than at home (Aix, Tölz, Hall in Upper Austria, Wiesbaden, etc.). These resorts are also suitable for after-treatment or for repeated courses of treatment, but the treatment by drugs is always much more important than the baths.

[Ehrlich has lately endeavored to find some substance that would have a special action in destroying the spirochæte pallida without harming the general organism. He has found dioxid-amido-arseno-benzol ("salvarsan," "606") the most efficacious remedy up to the present time. This he gives in a single dose—*therapia magna sterilisans*—which he claims destroys the syphilitic germ. It should be injected into the muscles or the veins in doses of gr. vij to viij (gm. 0.5 to 0.6) for men, or gr. vj (gm. 0.4) for women. It has given good results in all stages of syphilis, and in a number of cases the Wassermann reaction has changed from positive to negative. Cases of cure or amelioration have been reported from this remedy in severe forms of cerebral syphilis, but it does not seem to be of benefit in metasyphilitic disease, tabes, and general paralysis. It should be given very cautiously, if at all, in cases where the eyes are affected or the heart is weak. The injection may be repeated in a few weeks, especially in cases where the Wassermann reaction remains positive.—K.]

In addition to the specific treatment, a symptomatic treatment is necessary in many cases. Narcotics, local applications to the head, electricity, baths, etc., are to be used according to the same rules and with the same object in view as in other chronic brain diseases. Frequently the ætiological treatment is markedly assisted by these measures.

CHAPTER IX

PROGRESSIVE GENERAL PARALYSIS OF THE INSANE

(Paralytic Dementia. Paretic Dementia. General Paresis)

Preliminary Remarks.—Although the description of mental diseases is not a part of the plan of this book, we must nevertheless make an exception of one disease of the sort—namely, the so-called progressive general paralysis of the insane, or paralytic dementia, which in medical parlance is often abbreviated into "paresis." We consider it advisable to make this exception, because a great part at least of the symptoms of general paralysis are purely of a physical nature, and also because a knowledge of this disease, which is so common and so fatal in its results, is of the greatest importance for the general practitioner.

We must thank the French alienists, Boyle (1822) and Calmeil (1826), for the first clinical descriptions of general paralysis, by which it was more sharply differentiated than previously from the diseases which resemble it. A more accurate knowledge of the different symptoms, and the anatomical changes to which the morbid symptoms must be referred, has, however, only of late years been rendered possible by the introduction of better methods of investigation. Accordingly, we must now say that general paralysis is a disease which may attack the most diverse portions of the whole central nervous system [1]—the brain and spinal cord—at the same time or successively, although we can, of course, make out certain rules as to the predisposition of individual portions to disease, and as to the order of the symptoms. General paralysis begins most frequently in those regions of the cerebrum which have an immediate relation to the regular course of the psychical and certain psychomotor processes. Mental and motor symptoms accordingly form the introductory features of the disease in most cases. More extensive regions of the central nervous system are gradually involved in the morbid process, which goes hand in hand with a progressive degeneration of all the higher intellectual faculties, while at the same time many physical disturbances dependent upon nervous changes constantly increase.

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[1] At present hardly anything is known in regard to a primary implication of the peripheral nerves in the general process. The principle of such an implication would be readily understood in view of the almost regular occurrence of degeneration of the peripheral nerves in tabes.

Ætiology.—General paralysis is a common disease, and apparently demands a heavier quota from the better and more highly educated classes than from the lower classes. We may assume that, on the average, one tenth of all patients committed to the insane asylums are general paralytics. In most patients the beginning of the disease falls in the period between the thirtieth and fiftieth year. The disease is much rarer in advanced life. In young people under twenty it has hardly ever been observed. [Several hundred cases of juvenile general paralysis have been reported, almost always in victims of hereditary syphilis.—K.] There is no doubt that the male sex is much more frequently affected than the female, but the number of cases of general paralysis among women is not very small. [In juvenile paresis the sexes are about equally affected.—K.]

The cause of general paresis is always to be looked for in a previous syphilitic infection. Paresis, like tabes dorsalis (*q. v.*), is one of the metasyphilitic affections. Such an infection can be made out in at least seventy-five per cent of all cases. In this regard precisely the same conditions exist, and, of course, the same difficulties in the interpretation of this relation are to be considered, as we have previously mentioned in the account of the dependence of tabes dorsalis on syphilis (see page 333), a circumstance which again is not without significance, since the most intimate points of connection are to be found between tabes and general paralysis (*vide infra*). If we consider that general paralysis depends upon a previous syphilis, we have an easy explanation of most of the other peculiarities in the onset of the disease, especially the above-mentioned influence of age and sex, the decidedly common occurrence of the disease in persons in certain callings—such as artists and officers—the frequency of the disease in large cities, its rarer occurrence in the country, etc.

Besides the ætiological factors named, which, in our opinion, are alone of consequence, all other "causes" of general paralysis may well be regarded as merely predisposing. Mental overexertion has the greatest significance, especially if it be associated with psychical irritation. In merchants, civil officers, etc., who suffer from general paralysis, such a previous overexertion can often be made out. In some cases injury to the head or insolation is claimed to be the cause. Hereditary predisposition to nervous diseases plays perhaps a certain part in the origin of general paralysis, but by no means a very large one.

Clinical History.—General paralysis usually begins so slowly and gradually that a definite date for its beginning can hardly ever be given. In addition, it is often clear, at a time when the disease is already fully developed, that certain early symptoms, whose nature was at first not correctly recognized, ought to have been regarded as the initial symptoms.

The first symptoms of the disease in the psychical domain usually consist of the gradual appearance of a change in the whole nature and in the mental individuality of the patient, wherein, however, the mental disturbance usually shows from the start the character of weakness—that is, of a lessened capability of mental exertion. The patient's ordinary mental work no longer goes on as easily as before. His memory is uncertain, and there are marked forgetfulness and inattentiveness, which were previously quite impossible for him to exhibit. The patient is often disorderly in his dress, and violates the ordinary social rules of decency and morality. Since his judgment as to the value and significance of things is uncertain, he commits purposeless actions, wastes money, commits crimes, is dissolute, etc. In these respects, too, the increasing mental dullness often appears, since the patient becomes incapable of any higher intellectual, æsthetic enjoyment, and since the sway of nobler feeling finally becomes weak, and unable to exert any lasting influence upon his actions. Besides all these signs of beginning mental weakness, we often notice, on the other hand, an abnormal irritability. The patient easily becomes agitated, or gets angry; but these moods rapidly pass away without leaving a lasting impression. We easily understand how this change in the whole personality of the patient must distress and alarm his family, since the relatives at first cannot understand at all why he is now "so different from what he was."

In the first period of the disease a subjective feeling of illness is frequently present. The patient himself notices that his mental capacity, especially his memory, is diminished, and he very often becomes extremely anxious on this account. It frequently happens that certain subjective sensations are also noticed, a feeling of confusion in the head, pressure in the head, dizziness, rheumatoid pains, etc. The sleep is disturbed, as a rule, and also the appetite and the digestion. If such a patient comes to the physician with his complaints, it unfortunately only too often happens that he is in the beginning regarded as "neurasthenic," and is treated accordingly.

Careful observation, however, may usually even now discover the disease with certainty. The beginning mental disturbance is usually more apparent to the family than to the physician, who has not known the patient before and who sees him only cursorily, but it is apt to become manifest on a somewhat more searching examination of the patient. We generally succeed best by making the patient reckon; he often makes the greatest mistakes in simple examples in multiplication—especially does he fail in mental arithmetic, etc.

Certain motor symptoms, however, which usually come on in the early stages of the disease are of the greatest diagnostic significance, especially peculiar disturbances of speech and handwriting. The paralytic disturbance of speech shows itself first in the form of stumbling over syllables, or literal ataxia. The individual sound (in distinction from bulbar paralysis) can be pronounced quite correctly, but the combination of different sounds in the whole word causes increasing difficulties. It is a good plan, in order to recognize the first beginnings of this symptom, to have the patient pronounce a few difficult words, such as "third riding artillery brigade," "representative government," "initiative," "electricity," etc. We often hear "artralleriry" instead of "artillery," and like blunders. In the later stages of the disease the speech is sometimes almost wholly incomprehensible. We also observe other more complicated aphasic disturbances, such as paraphasia, persistent repetition of the same word, etc. In such cases the patients are sometimes no longer able to read any sentence correctly. They at times put in entirely different words, so as to make utter nonsense, but they do not notice it themselves. The abnormal associated movements of the facial muscles on speaking are also often to be observed, and are very characteristic. The voice of general paralytics often loses its power of modulation and becomes weak and rough—symptoms which depend upon a defective innervation of the vocal cords. The change in the handwriting, to be observed in general paralytics, is even more characteristic than the disturbance of speech (see Fig. 210). This is at first purely of a motor nature; the letters are uncertain, irregular, and tremulous. A psychical factor, however, also shows itself; single letters are omitted, the dot on the *i* and the marks of punctuation are forgotten, the patient ceases to keep on the lines, or leave a free margin, etc. As the disease advances, the disturbance in the handwriting gradually increases, so that the writing may finally become wholly illegible, and may consist merely of senseless scratches.

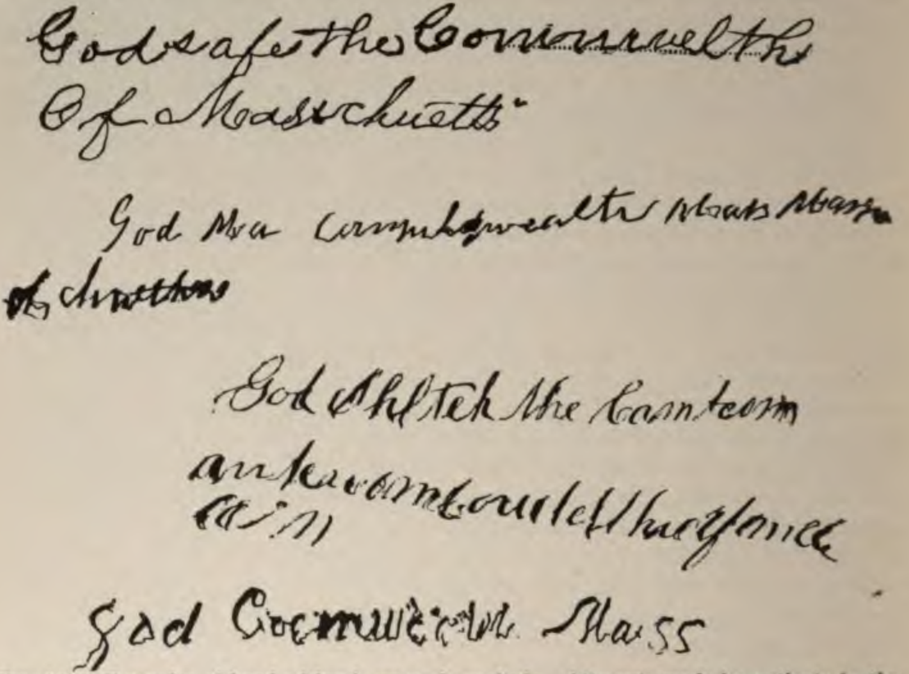
Godsafe the Commonwelth
Of Masschuetts

FIG. 210.—Examples of handwriting in general paralysis. Attempts made by patients in the Danvers Insane Hospital to write "God save the Commonwealth of Massachusetts." Besides the motor disturbance, the frequent omission of certain letters will be noticed, e. g., "Masschuetts" for "Massachusetts." In the last two the handwriting is almost wholly illegible.

Besides the changes in the speech and the handwriting, which we have just briefly described, other physical disturbances are often quite early symptoms, and prove in how many parts of the nervous system at once the disease may begin its work of destruction. The condition of the pupils especially is of importance in diagnosis. They are often unequal, and also show a reflex immobility (see page 349) in a large number of cases, especially in those in which other tabetic symptoms develop (*vide infra*). Transitory ocular paralyses are at times early symptoms. Not infrequently we find quite early changes in the tendon reflexes, very often a decided increase of the patellar reflex, or, in other cases, absence of the patellar reflex, which latter has a much more positive value in diagnosis—it is almost invariably a tabetic symptom (*vide infra*).

The association of reflex immobility of the pupils with an increase of the patellar reflex is not uncommon. In the distribution of the sensory nerves we may mention as repeatedly observed symptoms, neuralgia, attacks of migraine, and finally optic atrophy, the latter usually as one symptom of a coexisting tabes.

The changes in the cerebro-spinal fluid are entirely similar to those found in tabes (*cf.* page 355), viz., increase of lymphocytes and increase in the amount of albumen. The Wassermann-Plaut reaction is almost always positive with the blood serum as well as with the cerebro-spinal fluid, a further proof of the syphilitic origin of paresis. Under certain circumstances, all these conditions are also of great diagnostic value.

We often say that those cases belong to the "classical form" of general paralysis where an initial "stage of depression" with a melancholy tendency is followed by a second stage of "maniacal exaltation." This is the stage where the delusions, which are already quite pronounced, assume more and more the character of "grand ideas," and thus exhibit the "delusions of grandeur" which have for a long time been generally regarded as ominous. The first signs are often found in the patient's statements that he is now much better, that he is "very well," that he feels "very strong," etc. These delusions, however, often assume gradually a more exaggerated form—the patient considers himself enormously rich, he owns thousands of palaces, millions of dollars, has made the greatest inventions, considers himself the Emperor Napoleon, Christ, a "higher God," etc. Any judgment as to the absurdity of these ideas, and as to the sad contrast between his claims and the reality, has already become impossible to him; but, of course, there are even now occasional remissions in this condition, when the patient is clearer and recognizes temporarily the morbid character of his delusions.

We must not think, however, by any means, that the delusion of grandeur is necessarily an invariable symptom in general paralysis. In many cases (the so-called "depressive" form of general paralysis) the initial melancholic-hypochondriacal condition continues. The delusions that appear possess the same coloring: the patient claims that he can no longer eat, that he is poisoned, that he has lost his head or an arm, that he is very small ("*delire micromaniaque*"), etc. Sometimes acute and severe conditions of anxiety come on. In other cases, again (the agitated or maniacal form of general paralysis), there are states of violent excitement, in which the patient raves loudly, cries, and tries to destroy whatever falls in his way. Such states sometimes alternate with delusions of grandeur. Finally, we see cases of patients—and this is the most frequent form—who, in their mental relations, present simply the symptoms of a mental enfeeblement gradually increasing to complete dementia, without ever showing, in any notable form, states of excitement, the development of delusions, etc.

While the intellectual life progresses to utter ruin in the ways just stated, the physical disturbances of the disease, as a rule, gradually advance from bad to worse. In many cases ataxia of the extremities develops, and also loss of sensibility and vesical disturbances—in short, the symptoms of tabes. In these cases the tendon reflexes are almost always lost, and the pupils are often immobile. In other rarer cases, however, there is actual paralysis, first in the lower and then in the upper extremities. In these cases the tendon reflexes are often increased, so that the picture of "spastic paralysis" develops. Again, in other cases, bulbar symptoms appear, such as disturbances in swallowing, or masticatory paralysis, and also ocular paralyses, usually as a part of the tabetic symptom-complex, etc.

Peculiar attacks, which are among the commonest and most characteristic symptoms of general paralysis, are, however, of special interest, and sometimes even of marked importance in diagnosis. These "paralytic attacks" in their milder degrees sometimes appear even in comparatively early stages of the disease. Then they usually consist of attacks of vertigo, an obscuring of consciousness, or even a loss of consciousness, coming on quite suddenly, and lasting from a few minutes to half an hour or more, and they are not infrequently associated with mild hemiplegic or monoplegic symptoms. We very often see, besides the vertigo, a temporary feeling of weakness in the right arm, associated with a marked aphasic disturbance of speech. To this there is often added some slight twitching in the affected extremities or in the face. In the further course of the disease the attacks usually increase, and are termed apoplectiform or epileptiform paralytic attacks, according as the conditions of paralysis or spasm predominate. The epileptiform attacks may often be repeated with great frequency—thirty or forty attacks a day or more—during which time the patient remains in an unconscious state. If the patient gradually returns to consciousness, sometimes only after a week or two, we very often see, as a result of such severe attacks, a permanent impairment of the general condition, an increase of the dementia, etc.

The other organs, apart from the nervous system, are only secondarily implicated in the morbid process. However, the not infrequent association of paresis with syphilitic or metasyphilitic vascular affections (arteriosclerosis, aneurism of the aorta, etc.) is deserving of attention.

The temperature, as a rule, is approximately normal, or often somewhat subnormal, but very marked changes in the temperature occur in connection with the paralytic attacks—sometimes elevations and sometimes very deep declines.

The whole duration of the disease is in some cases only a few months (the "galloping" form of general paralysis), usually it is two or three years, and sometimes much more. The most rapidly fatal form is that in which there is very soon a marked emaciation and a rapid loss of strength as a result of the sleeplessness, the constant unrest, and the refusal of food. In other cases death ensues from the gradual and general loss of strength, or in a paralytic attack; or, finally, as is frequently the case, from the onset of secondary conditions, such as severe bedsores, pyelocystitis, tuberculosis, intestinal diseases, etc.

Pathological Anatomy and Nature of the Disease.—Considering the great difficulty of an accurate microscopic examination of the brain, it is not strange that our knowledge of the pathological anatomy of general paralysis is still very defective. If we except occasional immaterial changes in the skull, such as hyperostoses and the like, or in the meninges, such as hematoma of the dura or secondary thickening of the pia over atrophied portions of the brain,[1] the first striking and at any rate most important anomaly seems to be the atrophy of the brain, which affects chiefly the anterior half, especially the frontal lobes. In this region the convolutions are very much diminished and the fissures are wide; the weight of the anterior portion of the brain may be reduced to one fourth or one third of the normal. If we examine the convolutions microscopically we find that the diminution of the whole organ depends chiefly upon a loss of nervous elements. The cortex usually shows the greatest changes. The changes in the nervous elements themselves are the most important, and consist essentially of a degenerative atrophy of the ganglion cells and nerve fibers. We must mention especially that in the cortex of the frontal lobes, especially marked in the straight convolution and in the island of Reil, and also in other portions, we can make out with certainty, by the aid of good methods of examination, a very considerable loss of the fine medullary nerve fibers, mainly those which run parallel with the surface, and hence are termed "association fibers" (Tuczek); but signs of degeneration and atrophy are always to be seen in the ganglion cells themselves. In addition to this, marked changes are also found in the neuroglia and in the vascular and lymphatic systems. Numerous details, which have been recently determined (Nissl, Alzheimer) cannot be discussed here. The diffuse infiltration of the sheaths of the lymphatic vessels with plasma cells and lymphocytes is the most characteristic change in paresis. At any rate, with Tuczek, Wernicke, and others, I am entirely of the opinion that we have to do with a primary process of degenerative atrophy of the nerve fibers and nerve cells, to which the increase of the connective tissue and the so-called "inflammatory" and infiltrative changes are only a secondary addition.

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[1] [An adhesion of the pia mater to the brain substance is very characteristic. This adhesion may be so firm that bits of the cortex are torn away on endeavoring to separate the pia from the brain.—K.]

The anatomical affection in general paralysis, moreover, is by no means limited to the cerebral cortex. We can almost always make out marked changes in the nerve tissue and the interstitial substance in the deeper parts, also, in the white substance, and the central ganglia. The coexisting changes in the spinal cord, first accurately described by Westphal, and since then recognized as almost constant, are of especial interest. They usually consist of fascicular systemic degeneration of the lateral columns (the pyramidal tract) or the posterior columns. A large part of the physical disturbances of general paralytics, such as tabetic symptoms or spastic paralysis (*vide supra*), are certainly due not to the cerebral disease, but to these accompanying changes in the spinal cord.

Accordingly, we believe that, on the basis of our present knowledge, we can best conceive the nature of general paralysis in the following way: By the action of certain toxic substances depending on a previous syphilitic infection, there is a gradually progressive destruction of nerve tissue in the most diverse portions of the nervous system. The clinical symptoms must naturally differ according to the significance and function of the affected fibers or cells. As a rule certain cortical regions of the cerebrum are first diseased. The disturbances of speech are probably dependent upon the loss of fibers in the left island of Reil, the disturbances of intelligence upon the destruction of fibers in the cortex, particularly in the frontal lobe. We may also make out corresponding anatomical changes, either cerebral or spinal, as an explanation of the later motor, tabetic, and other symptoms; but in many cases the order in which the different sections are affected varies considerably. We have seen in a previous section (see page 355), that the whole process may begin with a spinal disease, especially tabes dorsalis, to which the paralysis is "added" later; but we must understand that the two conditions are wholly analogous to and coördinate with each other. Both are parts of the same degenerative process, which can accomplish its work of destruction in the most diverse regions of the nervous system.

No coarse anatomical lesions can be made out, as a rule, to explain the paralytic attacks, but it is very probable that they depend, at least in great part, upon the changes in the motor central convolutions.

Diagnosis.—Since the diagnosis of beginning general paralysis is of the greatest practical importance, we will once more mention briefly all those symptoms which are especially to be considered in diagnosis: Striking alteration in the nature and the behavior, rapid and motiveless change in the disposition, disturbances of memory, loss of intelligence (failure in reckoning, etc.), the characteristic changes in the speech and handwriting, and finally the coexisting somatic symptoms—inequality of the pupils, immobility of the pupils, loss, or [more frequently] increase of the tendon reflexes, and mild paralytic attacks, such as vertigo, disturbance of speech, temporary disturbance of motion in one arm, etc. [Examination of the cerebro-spinal fluid, obtained by lumbar puncture, is often of great assistance in the diagnosis.—K.]

We would also mention, as especially common and disastrous mistakes, that the symptoms of general paralysis are often misunderstood at first, and are regarded as the signs of immorality, the failure of the sense of duty, etc. It also frequently happens that general paralysis is at first regarded as simple neurasthenia or hypochondriasis, and treated accordingly.

As a rule, general paralysis can be certainly distinguished from other organic nervous diseases by careful attention; but of course we must add that in some cases cerebral tumors, gummatous processes, and especially certain cases of multiple sclerosis, may show a type of disease very like general paralysis. Simple senile dementia, due to arteriosclerosis, and certain forms of chronic alcoholism may also be confused with general paralysis.

Prognosis.—The prognosis of general paralysis, like that of all chronic degenerative conditions of the central nervous system, is wholly unfavorable. At present we know of only a small and decreasing number of (doubtful) recoveries, but there are many cases where there is a temporary improvement in the condition, a "remission," sometimes of a considerable degree and lasting a long time. [Dana has recently reported several cases of apparent recovery after the use of mercury in the early stages of the disease.—K.] The earlier the patient comes under proper care and treatment the sooner may we hope for such a favorable turn. Of course, as we have said, relapses of the disease almost always come on later. Those cases especially are to be regarded as unfavorable in which other physical symptoms, especially of a spinal nature, soon set in, and in which the whole nutrition of the body rapidly suffers.

Treatment.—As soon as the disease is recognized, the first and imperatively necessary injunction must be to remove the patient from all physical and intellectual exertion as well as from all mental excitement. The patient must therefore, if possible, withdraw from business, which up to that time he may have tried to carry on. His methods of life and his diet must be regulated, and every excess must be forbidden. For the cases which even at first are associated with states of great mental excitement, the commitment to a proper asylum is often most urgently to be recommended, while for cases that during their course show simple mental weakness, care at home is often sufficient. Only by a timely recognition of the disease and by adopting the appropriate measures can the patient's relatives be spared the many disagreeable experiences which would otherwise undoubtedly ensue.

In regard to the treatment of the disease itself, we should advise inunction with mercurial ointment, especially if we can discover a previous syphilitic infection. As a rule we ought not to expect much success from this any more than in tabes (see page 358), but we may perhaps check the advance of the disease. We should therefore try the antisyphilitic treatment chiefly in the initial stages of the disease, and also in the cases where the suspicion that there is also gummatous syphilis is justified. The preparations of iodin are frequently prescribed, either alone or in combination with bromids.

Furthermore, we should try tepid baths, with cool sponging, half baths, with brief douches, and also a cautious application of electricity (galvanization of the head and spinal cord), and prescribe internal remedies, especially ergotin. We need not go more fully here into the numerous symptomatic details.

CHAPTER X

CHRONIC HYDROCEPHALUS

(*Meningitis serosa ventriculorum*)

Ætiology and Pathology.—Repeated mention has been made in preceding chapters of the occurrence of dropsy in the ventricles as a sequel to other cerebral diseases, such as meningitis and tumors. Besides this "secondary hydrocephalus," a collection of fluid in the ventricles may be a symptom of an apparently idiopathic primary disease. This is observed most of all in the newborn, or at least in young children.

Little is known with certainty about the causes of chronic hydrocephalus. The assumption is very frequently made that the condition is the result of an inflammation of the ependyma of the ventricle, which itself occurs either before birth or very soon after, but the autopsy often fails to support this idea. Our knowledge is equally uncertain as to the existence of a stasis due to mechanical obstruction (obliteration of the foramen of Magendie, etc.). Syphilis, drunkenness in the progenitors, etc., have been regarded as predisposing causes; whether justly or not is uncertain. It has been repeatedly observed that the disease has attacked several children of a single family.

The most important physical sign of hydrocephalus in children is enlargement of the head. The circumference of the skull may even in the first year of life be 60 to 80 cm. Usually the frontal bones and the parietal eminences are especially prominent. The cranium becomes gradually almost as thin and translucent as paper. The fontanelles and sutures gape widely. The brain is flattened out, so as to seem almost like a bag, filled with the hydrocephalic fluid. In well-marked cases the entire thickness of the hemispheres is frequently not more than an inch. The space within, containing the serous effusion, represents the enormously distended ventricles, particularly the lateral ventricles, although the third and fourth ventricles are quite often distended also. The walls of the ventricles are often strewn with minute granulations, or they present a reticular hypertrophy. The hydrocephalic fluid usually has the appearance of colorless serum, and contains a very slight amount of albumen, if any. The specific gravity is about 1.004 to 1.006. The amount of fluid may be a quart or more; but, of course, there is a great variation in this respect in different cases.

Congenital hydrocephalus is often associated with other peculiarities or defects in the structure of the brain, which we cannot mention here.

Clinical History.—Sometimes a child is born with hydrocephalus so far developed as to occasion dystocia. Usually, however, the parents notice nothing peculiar about the child for some weeks. Then they are alarmed by the gradual swelling of the head. As a basis for determining abnormal size, we may mention that under normal conditions the circumference of the head at birth is about 30 to 40 cm., at the end of a year about 45 cm., and from that age to puberty there is a gradual approach to a circumference of about 50 cm. The possible dimensions in chronic hydrocephalus have been already stated. The increase in circumference is often quite rapid, amounting in two or three weeks to 1 or 2 cm. Usually the swelling is tolerably symmetrical, but sometimes the greater increase is in the antero-posterior diameter, making the skull dolichocephalic. At times the rate of expansion may be particularly rapid, and then at other times it may seem to be suspended. That the fontanelles and sutures remain widely open has already been mentioned; sometimes it is even possible to get fluctuation through them. An intravascular murmur can now and then be heard in the head, but it has no great importance with regard to diagnosis. The veins are often so greatly distended as to form a bluish network underneath the scalp. The face remains small, in striking contrast with the great, heavy cranial portion of the head. The head almost always hangs over forward, from its own weight. The eyes generally look down, partly because the roof of the orbit is depressed, and partly because of impairment of the nervous supply to the motores oculi.

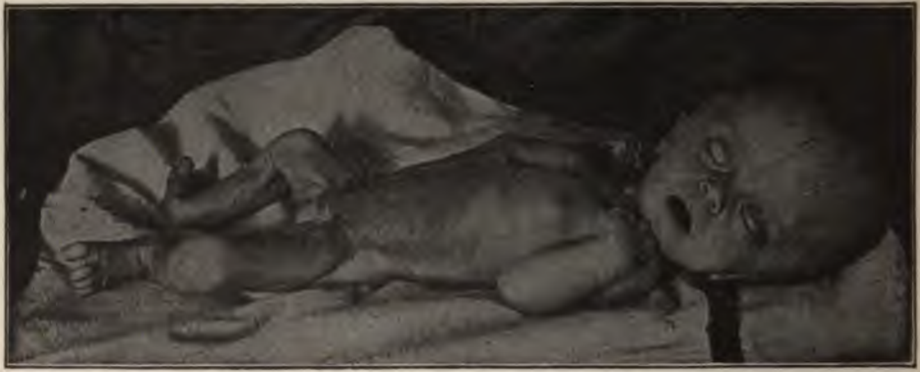

FIG. 211.—Chronic hydrocephalus (after SCHLOSSMANN).

A very important symptom is the defective intellectual development of hydrocephalic children. They cannot learn to talk well, if at all. If they play, it is in a silly manner. They cannot concentrate their attention upon anything, and they are heedless and dirty. It must, however, be mentioned that sometimes, in spite of considerable hydrocephalus, the patient now and then evinces an unexpected activity of mind; thus, he gradually becomes able to distinguish the different objects and individuals about him.

There is almost invariably motor disturbance also. The legs, more rarely the arms, are decidedly paretic, or there may even be complete paraplegia. There are usually spastic symptoms and increased tendon reflexes. Few patients learn to walk or stand alone. The arms seldom present any great paresis, but their movements often betray an awkwardness and uncertainty suggestive of ataxia. It is noteworthy that sensation almost always remains intact; at least the patient reacts vigorously to the prick of a pin, etc. Of the special senses, sight is most frequently affected; choked disc and atrophy of the optic nerve have been observed repeatedly. Symptoms of motor irritation are of very frequent occurrence, such as general convulsions and spasm of the glottis. General nutrition is pretty well maintained in many cases, but, as a rule, hydrocephalic children are atrophic and ill developed.

The chronic hydrocephalus of children almost always terminates unfavorably. Only a few patients survive the fifth year, although now and then striking exceptions occur. Death is generally the result of marasmus, or a convulsive seizure may prove fatal. The possibility of recovery has not yet been demonstrated. The progress of the disease may, however, be arrested, and the child continue for years *in statu quo*. A certain amount of symptomatic relief has been repeatedly observed to take place spontaneously, by the cerebro-spinal fluid forcing its way to the outer air. This discharge generally takes place into the nasal cavity through the cribriform plate of the ethmoid, so that an evacuation of cerebral fluid occurs from the nose; of course, mostly only drop by drop, but occasionally also more abundantly. This same phenomenon, moreover, has also been observed in cases of brain tumor, and, in isolated instances, even in apparently healthy individuals.

We have become better acquainted with the acute or chronic, apparently idiopathic, hydrocephalus of adults only within recent times. Quincke has assumed as its cause a chronic inflammatory disease of the ependyma of the ventricle (so-called meningitis serosa ventriculorum). The clinical symptoms of those cases, which run an acute course, are very similar to those of suppurative meningitis, but the symptoms are less violent, as a rule, and the termination is more favorable. Occasionally the acute initial stage is followed by a protracted chronic stage, which is subject to many fluctuations in intensity. Other cases pursue a more chronic course from the onset, and present a clinical picture which must necessarily arouse grave suspicion of brain tumor (headache, vertigo, choked disc or atrophy of the optic nerves, motor disturbances, etc.). Some of these cases show a marked improvement later on, but they may also terminate fatally, and only the autopsy will show the diagnosis (generally of "brain tumor") to be incorrect. The results of lumbar puncture are sometimes of importance in respect to differential diagnosis—evacuation of the cerebro-spinal fluid under high pressure, and marked increase of the leucocytes.

Personally, I am inclined to the opinion that most, though by no means all, of the cases belonging to this category should be included under disseminated encephalitis, and that the hydrocephalus is a secondary manifestation, just as exudative pleurisy, to use a common example, is secondary to a primary affection of the lung or lymphatic glands. By careful inquiry a fairly acute onset can also be frequently demonstrated in the chronic cases. It is true that I cannot offer any anatomical proof of my views, but nevertheless I desire here to express briefly my opinions, which are based on considerations stated elsewhere (*vide* page 541).

Diagnosis.—A pronounced case of congenital hydrocephalus can be recognized without difficulty, inasmuch as the excessive size of the head betrays the disease upon the first glance. Less extreme cases may indeed be somewhat obscure, and we have especially to avoid confounding the condition in question with the macrocephalic, but generally markedly square, rachitic enlargement of the skull. We should always, therefore, take into consideration the intellectual powers, the presence or absence of motor disturbances, and other similar symptoms, as well as the cranial peculiarities. In the hydrocephalus of adults there is often no enlargement whatever, so that a diagnosis can hardly ever be made positively.

Treatment.—Thus far, no remedy has been successful in chronic hydrocephalus. The following may be tried: Applications of mercurial ointment and of tincture of iodin to the scalp, methodical compression of the skull, and iodid of potassium internally. The hydrocephalic fluid has often been partially drawn off by tapping and other operative procedures, but only rarely with permanent success, and usually with merely temporary benefit, if any. Quincke was the first to suggest drawing off the hydrocephalic fluid by lumbar puncture. In most cases we confine ourselves to purely symptomatic treatment, and to recommending intelligent care of the child.

In acute hydrocephalus, the so-called serous meningitis of adults, lumbar puncture is also to be considered the chief therapeutic measure. Quincke, moreover, highly recommends inunctions of mercurial ointment, and, in grave cases, also irritating ointments (antimonial ointment) applied to the shaven head. Daily, for three or four days, a piece of antimonial ointment [unguentum antimonii et potassii tartratis] the size of a pea is rubbed into the skin, and thus a severe "derivative" dermatitis is produced.

CHAPTER XI

MÉNIÈRE'S DISEASE

(*Vertigo ab aure læsa. Labyrinthine Vertigo*)

In 1861, Ménière, a French physician, first called attention to a peculiar affection which may be apparently primary or may result from chronic aural disease, and which is characterized mainly by excessive vertigo and loud tinnitus aurium. At present we know that we have to deal invariably with an affection of the vestibular apparatus (semicircular canals of the labyrinth), though the anatomical changes and the corresponding entire course of the disease may vary greatly. It is obvious that Ménière's symptom-complex may be closely related to other forms of vestibular vertigo, such as are observed in cerebellar disease and similar affections. The differentiation of true Ménière's disease is, however, afforded by the localization of the fundamental disease in the internal ear.

Symptomatology.—The clinical symptoms which constitute Ménière's disease are the following: (1) *Vertigo.* We generally have to deal with true rotary vertigo—i. e., a sensation as if the body itself were being turned about in a circle, or (when the eyes are open) as if external objects were moving in a circle. The direction of rotation is either always the same or it varies. In other cases the apparent movements are irregular. The patients have the sensation of being thrown back and forth, of falling, etc. These symptoms of vertigo persist even during absolute rest. Consciousness as such is, as a rule, not clouded. (2) Walking and standing become seriously affected by the vertigo. The gait is uncertain and staggering, and, occasionally, entirely impossible (vestibular ataxia). At the sudden onset of an attack of Ménière's disease the patients sometimes fall to the ground and sustain serious injuries. (3) Almost invariably the vertigo is associated with pronounced nausea, which frequently culminates in violent retching and vomiting. Numerous loose movements of the bowels are also not uncommon. (4) On careful examination oculomotor symptoms can generally be found—nystagmus and deviation of the eyes. These phenomena are evidently associated with the physiological function of the vestibular apparatus. (5) Tinnitus aurium and deafness are justly considered as almost constant, and, as regards diagnosis, decisive symptoms. As a matter of fact a pronounced tinnitus aurium is almost invariably associated with Ménière's vertigo, and is described by the patients as whistling, roaring, ringing of bells, hammering, etc. However, we must bear in mind that tinnitus aurium is an acoustic symptom, and consequently we can understand that it is found in almost all affections of the internal ear, just the same as deafness. There are, however, rare cases of Ménière's disease in which the acoustic nerve symptoms are entirely absent, or at least are not at all pronounced. (6) Finally, we must mention, as frequent concomitant symptoms, sweating, nervous chills, accelerated pulse (frequently slightly irregular, rarely diminished in frequency), a feeling of heaviness in the head, etc.

This symptom-complex, as we have previously mentioned, appears under widely differing conditions. Most frequently Ménière's disease follows chronic ear diseases, whether they originated in the middle ear or primarily affected the internal ear (chronic suppurative otitis, otosclerosis, etc.). Frequently the patients suffer constantly from slight tinnitus aurium, with or without difficulty in hearing, and are subject to attacks of Ménière's vertigo only from time to time. In other cases the symptoms appear acutely or almost in an apoplectiform manner in persons who previously had perfectly healthy ears. Cases of this sort, especially when they are associated with slight loss of consciousness at the onset, may very readily at first be mistaken for true cerebral apoplexy. In a very short time total deafness, very severe vertigo, staggering gait, and marked tinnitus aurium may supervene. The symptoms gradually diminish in the course of time, but the recovery is scarcely ever complete. An acute hemorrhagic inflammation of the labyrinth is assumed to be the anatomical cause of this apoplectiform Ménière's disease. Hemorrhages into the labyrinth (in arteriosclerosis) can also occur. Frankl-Hochwart describes a combination of acute Ménière's disease with facial paralysis, and interprets such cases as "polyneuritis cerebralis menieriformis."

Not infrequently Ménière's disease occurs after severe injuries to the skull (traumatic hemorrhage into the labyrinth, fractures of the petrous portion of the temporal bone). Furthermore, it has been repeatedly observed in the clinical picture of other diseases, viz., syphilis, tabes dorsalis, leukæmia, acute infectious diseases (meningitis, typhoid, mumps). Finally, marked attacks of Ménière's disease have also been observed in cases in which the subsequent favorable course of the disease indicated that we were dealing only with slight functional disturbances. Some attacks seem to be related to migraine and scintillating scotoma; others, to neurasthenia and possibly, also, to epilepsy. Lastly, it is to be noted that the well-known symptoms of acute quinin, salicylic acid, and, possibly, too, nicotin poisoning must also in part be regarded as vestibular symptoms.

Diagnosis.—The diagnosis of Ménière's disease can generally be definitely made if we have a thorough knowledge of the symptom-complex, and examine the patient carefully. The prognosis depends upon the particular form of the disease in the case in question.

Treatment.—Treatment may be divided into (1) special otological treatment of the underlying aural disease; (2) general treatment, viz., rest, high altitude, baths, electro-therapeutics, etc. If there is a suspicion of syphilis, iodids and mercury should be administered; (3) symptomatic treatment—bromids, nux vomica, antipyrin, etc. Charcot claimed that the continuous use of quinin produced an improvement, at least in many cases, and that occasionally a complete cure resulted. We prescribe 5 to 10 gr. (gm. 0.3 to 0.6) of quinin, divided in two or three doses, and give this remedy for several weeks at least. Personally, I must indeed confess that my results with quinin have not been very satisfactory in those appropriate cases in which it was tried. Occasionally continued galvanic treatment (anode to the ear, cathode to the nape of the neck) appeared to us to be of decided benefit. In grave cases lumbar puncture is said sometimes to produce good results.

V. NEUROSES WITHOUT KNOWN ANATOMICAL BASIS

CHAPTER I

THE SO-CALLED VASOMOTOR AND TROPHIC NEUROSES. ERYTHROMELALGIA. SYMMETRICAL GANGRENE. ACUTE ANGIONEUROTIC ŒDEMA. MYXŒDEMA. ACROMEGALY. [GIGANTISM. HYPEROSTOSIS CRANII.] SCLERODERMA. FACIAL HEMIATROPHY. HYDROPS ARTICULORUM INTERMITTENS. INJURIES OF THE CERVICAL SYMPATHETIC.

General Consideration of Vasomotor Disturbances.—Physiology, as is well known, distinguishes two varieties of vasomotor nerves—the vasoconstrictors and the vasodilators; but since experiments have detected the latter variety in only a few places—for example, in the chorda tympani, the nervi erigentes, and the sciatic—they have not acquired a very great significance in human pathology. We are at present much more disposed to refer every abnormal constriction of the vessels to an excitation, and every abnormal dilatation of the vessels to a paralysis of the vasoconstrictor nerves, although perhaps pathological conditions of excitation of the vasodilators may not be at all rare. In regard to the precise anatomical course of the vasomotor nerves, we must first state that vasomotor excitations may certainly proceed from the cerebrum, as is shown by the well-known symptoms of blushing and pallor from mental emotions. In experiments on dogs, Eulenburg and Landois have succeeded in producing a fall of temperature on the opposite side by irritating certain portions of the cortex in the immediate vicinity of the motor centers, and by extirpation of the same parts they have produced a rise in temperature. Furthermore, we know with certainty that there is an important vasomotor center in the medulla oblongata (in the region of the upper olivary body in rabbits), the excitation of which, directly or reflexly, is followed by an almost universal vascular constriction, and its destruction by an almost universal vascular dilatation. We must probably seek the further course of the vasomotor nerves very largely (or exclusively?) in the lateral columns, from which they pass out chiefly by the anterior roots. It is not known with certainty whether there is any decussation of the vasomotor fibers, or, if so, where it occurs. The larger part of the vasomotor nerves collect, at any rate, in the principal trunks of the sympathetic, from which, as is well known, the separate plexuses that surround the vessels arise. It is not improbable, however, that there is also in part a direct passage of vasomotor fibers from the cord into the peripheral nerves. In conclusion, we must mention that, according to Goltz's experiments, there are reflex vasomotor centers in the cord for the different parts of the body.

The clinical vasomotor symptoms are chiefly to be observed in the external skin. We distinguish them as follows:

1. *Symptoms of Vasomotor Paralysis.*—We conclude that there is a paralysis of the vasomotors if there is an abnormal redness of the skin. Such a redness is almost always associated with an objective and often a subjective feeling of an increase of temperature. Such conditions are observed either in connection with other nervous symptoms—as in fresh spinal or cerebral paralyses, and also very often in certain functional neuroses, such as hysteria and neurasthenia—or in the form of independent affections—the pure vasomotor neuroses, injuries of the cervical sympathetic, etc. Of course in these cases it is usually hard to decide whether there is really a vasomotor paralysis or irritation of the vasodilators. There are cases in which the only symptoms are a persistent or paroxysmal diffuse redness of the skin, especially of the head, associated with a great feeling of heat, with palpitation of the heart, strong pulsation of the arteries, anxiety, ringing in the ears, and sweating.

2. *Symptoms of Vasomotor Spasm.*—Spasm of the small vessels becomes apparent by a striking pallor and coolness of the skin. There is often, with this, a decided feeling of formication and stiffness in the affected parts, which may even increase to an actual feeling of pain. Such vasomotor spasms affect the hands especially, and form a chronic trouble that is not very rare. They are usually seen in people who are generally nervous and irritable, and sometimes also in washerwomen. A vascular spasm is sometimes seen in the ex-

tremities as one symptom of complicated paroxysms, such as nervous angina pectoris (*q. v.*), especially at the beginning of the paroxysm. A persistent spasm of the small arteries may give rise to considerable subsequent trophic disturbance. At least, the rare cases of so-called "spontaneous symmetrical gangrene" (*q. v.*), and also certain forms of scleroderma and some similar affections, are referred by many observers to a primary spasm of the vessels.

General Consideration of Trophic Disturbances.—We have much less information concerning the trophic nerves than we have concerning the vasomotor. As is well known, the controversy is still going on as to whether we have any right to assume the existence of special trophic nerves. The facts of clinical neurological observation frequently seem to compel the hypothesis of special trophic functions, although, of course, it is usually very hard to determine this point, because we can seldom wholly exclude the indirect influence of sensory and circulatory influences (*vide supra,* anæsthesia of the trigeminus). Recent observations on the significance of the so-called internal secretions have opened up entirely new lines of thought on the nature of trophic disturbances. It has been known for a long time that diseases of the thyroid gland (myxœdema, exophthalmic goiter), the hypophysis (acromegaly), the suprarenal glands (Addison's disease) cause remarkable morbid symptoms which are trophic in character. We shall probably learn additional facts bearing on these subjects which will tend to enlighten us regarding many morbid processes which up to the present time have proved to be rather enigmatical.

Among those symptoms which chiefly support the theory of the existence of specific trophic nervous influences, we have already learned to recognize the degenerative atrophy of the muscles and nerves. Various other sorts of trophic disturbances in the skin and the deeper parts (bones, joints) are seen in nervous diseases. Especially after wounds of the peripheral nerves we often notice a peculiar shining, smooth, atrophic condition of the skin—the "glossy skin" or "glossy fingers" of English authors. In other cases anomalies in the pigmentation of the skin seem to be connected with the nervous disturbances; thus, spots deprived of pigment (*vitiligo*) often develop as a result of severe neuralgias. We must also bring to mind here the appearance of changes in pigmentation from nervous causes, especially the ætiology of Addison's disease (*q. v.*) and the occurrence of the so-called nervous nævi. Among the severe neurotrophic disturbances of the skin many observers, especially Charcot, class the appearance of acute bedsores in many spinal and cerebral paralyses, but we have never been able to convince ourselves of the occurrence of a "neurotrophic decubitus," and we believe that every bedsore is due in the first instance to external injuries (pressure on the soft parts, uncleanliness, and the entrance of the bacteria of inflammation).

Besides the trophic disturbances in the skin, we often see analogous changes in the nails and hair in nervous patients. The nails become brittle and cracked, assume a darker color, and often show abnormal conditions of growth, abnormal curving, striation, thickening (*onychogryphosis*), etc. We also see at times a loss of the nails. A loss of hair is seen in frontal neuralgia, in certain forms of headache, and not infrequently as an apparently independent nervous disease (*alopecia*). A very rapid whitening of the hair after mental excitement is well known to have occurred in some cases.

Various disturbances of growth and nutrition also occur in the bones and joints, and are to be regarded as trophic disturbances.

The implication of the bones in atrophic processes is seen chiefly in progressive unilateral facial atrophy (*vide infra*). A retarded growth of bone in the affected extremities is also a symptom frequently seen in the spinal, and even in the cerebral, paralyses that develop in childhood. It is uncertain, however, how far this is due to the loss of direct trophic influences or to the indirect influence of the loss of motor functions. In conclusion, we not infrequently see very striking and apparently trophic disturbances in the bones and joints in tabes and syringomyelia (*q. v.*).

Erythromelalgia.—We should apparently class here this peculiar disease, which is seen most frequently in the feet, although it also occurs in the hands. Men seem to suffer from it more frequently than women. The symptoms are severe pain, especially in the toes and fingers, but sometimes in the ball of the foot, the heel, etc., and also swelling, redness, and marked pulsation of the vessels. In some cases the affected parts assume a very dark red or bluish-red color. We not infrequently see excessive sweating. [In some cases the condition seems due to neuritis; in others Weir Mitchell, who first described the disease, has found arteriosclerosis in the smaller vessels with no neuritis.—K.]

The condition described either comes on in paroxysms or is persistent, although varying in severity. On standing or walking, or when warm, the pain usually increases. Other nervous symptoms, such as headache, vertigo, weakness, etc., are sometimes associated with erythromelalgia. The course is almost always very tedious, and the prognosis therefore doubtful. The treatment consists in the use of electricity, cool baths, iodid of potassium, antipyrin, and similar remedies. [Mitchell suggests stretching or resection of the nerves.—K.] Compare the section on acroparæsthesia (page 186), which name has sometimes been given to apparently allied conditions.

Symmetrical Spontaneous Gangrene. Raynaud's Disease.—This disease, whose nature is still quite obscure, begins with pallor and coldness of the fingers ("regional ischæmia"), associated at first with sensations of crawling and numbness, and later with severe pain. After a short time the pallor changes to a bluish-red color ("regional cyanosis"), especially in the terminal phalanges, which sometimes progresses to the formation of gangrenous spots and vesicles. In severe cases a portion of the terminal phalanges is completely destroyed and thrown off by dry gangrene. The whole process may last for months. It runs its course without fever, but it is sometimes associated with quite severe general nervous disturbances. In rare cases spontaneous symmetrical gangrene has been observed in other parts of the body—for example, in the thighs, the buttocks, the ears, the nose, etc. Several attacks of the disease may sometimes follow one another, so that the whole duration of the trouble may last for years.

In regard to diagnosis, we must first be convinced that the disease comes on spontaneously. Extensive areas of gangrene of the skin have repeatedly been observed in hysteria, produced artificially by the patient by means of caustics (caustic potash, etc.). We must also think of diabetic gangrene, severe arteriosclerosis, syringomyelia, exophthalmic goiter, etc.

The treatment can be merely symptomatic. Wet applications, baths, cautious galvanization, and narcotics (morphin, chloral) when the pain is severe, are most employed.

Acute Angioneurotic Œdema (*Œdema cutis circumscriptum*).—This name has been given by Quincke, Strübing, and others to a disease characterized by the sudden onset of œdematous swellings in various parts of the body. These swellings usually disappear in a few hours, but they are very often repeated. Dangerous symptoms may arise if the œdema affect the pharynx or the entrance to the larynx. The patient's health otherwise is sometimes perfectly good, but in other cases it is more or less affected. Gastric disturbances especially (attacks of vomiting and gastralgia) have been observed at the same time in such patients. Acute angioneurotic œdema is manifestly closely allied to urticaria and erythema exsudativum.

Myxœdema.—We may here mention myxœdema (*cachexie pachydermique* of Charcot), which was first described in England by William Gull and Ord. The disease has taken its name from a peculiar thickening and swelling of the skin, which is most marked in the face, but which is sometimes seen in the extremities, the trunk, the tongue, and even in the internal organs. This swelling is not œdema, but it is due to the development of a sort of myxomatous new growth, rich in mucin, in the connective tissue. Other trophic disturbances usually coexist—atrophy of the teeth and nails, [spade-like hands, coarseness and] loss of hair, failure in the sweat secretion and consequent dryness of the skin, etc. Besides these there gradually develops a general physical and mental weakness, which may increase to great hebetude or even to complete dementia. Disturbances of the sensory functions may also occur. It is a fact of especial interest that we find quite commonly a diminution and even a complete atrophy of the thyroid gland. It is very probable that all the symptoms of the disease may arise from the failure of function of the thyroid gland. This hypothesis is confirmed by the repeated experiments of Kocher and others, that after complete extirpation of the thyroid in man almost the same symptoms ensue as in myxœdema (*cachexia strumipriva*). From this we must suppose that certain injurious substances accumulate in the body which are ordinarily rendered innocuous by the thyroid gland.

This theory has been materially supported by recent extremely interesting experience in the treatment of myxœdema. The physiologist Schiff made the remarkable observation that the symptoms of cachexia strumipriva did not appear in animals if the thyroid gland of another animal were transplanted into the abdominal cavity. Horsley, Bircher, and others hereupon made the first experiments in the treatment of myxœdema in man with extract of the thyroid gland (from sheep, calves, etc.). At first subcutaneous injections of thyroid extract were usually employed, but it was found that the internal administration of the substance of the gland or an extract thereof had an equally favorable influence. There are already very many positive observations which show that all the symptoms of myxœdema in man may be made to disappear completely in the course of a few months by the continued internal administration of the thyroid gland. The most convenient and certain form of administration is in tablets. Of course we must always be prepared for relapses. [Myxœdema is sometimes associated with exophthalmic goiter and rarely with acromegaly. See the chapter on Idiocy (page 701) for the consideration of cretinism and myxœdematous infantilism.—K.]

Acromegaly.—A "trophic disturbance" chiefly of the bones, but also involving the soft parts, forms the basis of that peculiar and rare disease recently termed acromegaly (P. Marie, Erb, etc.). The affection develops slowly in women and men, usually in youth or middle age. In women cessation of menstruation almost invariably precedes the onset of the disease. Besides the general symptoms of dullness, fatigue, and quite severe neuralgic or drawing, deep-seated pains in the head and the extremities, there gradually develops a striking increase in the size of the hands and feet, and thickness and plumpness of the face, due chiefly to enlargement of the nose, chin, and lips, which become puffed out to a considerable size. The increase in the lower part of the face (especially the lower jaw) in contrast to the normal cranium gives a very characteristic aspect to acromegaly. The hyperplasia of the lower jaw may reach so high a degree that the individual teeth are separated. In the hands and feet, the fingers and toes, and especially their terminal phalanges, become thicker and wider, so that we may speak of actual "paws." The hyperplasia not only affects the bones, but it makes the skin thick and wrinkled. A manifest kyphosis of the spine usually develops. Other noteworthy symptoms sometimes occur. Thus, we sometimes see marked glycosuria, an increased secretion of sweat, and particularly disturbances of vision, either hemianopsia [especially bitemporal hemianopsia.—K.] or amblyopia from atrophy of the optic nerves. These disturbances of vision are connected with the noteworthy fact that in most cases of acromegaly a tumor of the hypophysis cerebri develops, which presses directly on the nerves of the optic tract. A patient under our observation (Fig. 212) died with symptoms of a tumor at the base of the brain, and the autopsy

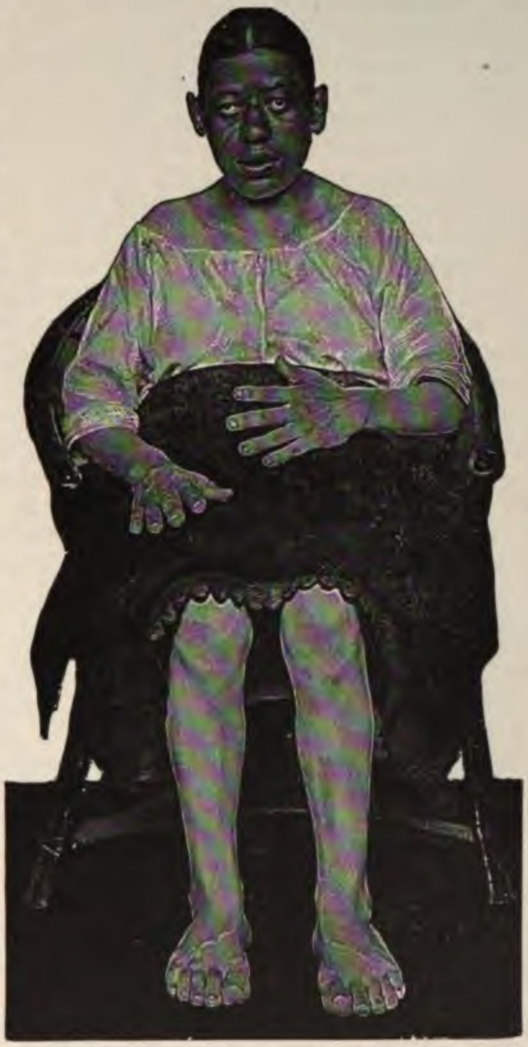

FIG. 212.—Acromegaly in a patient thirty years old. (Personal observation.)

showed a sarcomatous tumor of the hypophysis which had grown through the adjacent bony parts.

The diagnosis of acromegaly is not difficult. We must merely guard against confusing it with other conditions also leading to thickening of the bones (partial giant growth, arthritis and osteitis deformans, diffuse and localized hyperostoses, etc.). The so-called hypertrophic osteoarthropathy is also to be distinguished from acromegaly. This affection is seen especially in patients with chronic bronchitis, bronchiectasis, chronic tuberculosis, etc., and it also leads to a paw-like thickening of the fingers, but the changes in the lower jaw so very characteristic of acromegaly are absent, and also the other nervous symptoms, especially those of tumor of the hypophysis.

The treatment of acromegaly is, unfortunately, almost wholly unpromising. The administration of thyroid tablets and also of tablets of the hypophysis has been tried without much benefit.

[**Gigantism.**—The "giants" who exhibit themselves in circuses and museums are victims of a disease closely akin to acromegaly, if not identical with it. A study of giant skeletons has shown a marked enlargement of the sella turcica, the site of the hypophysis, and an enlargement of the hypophysis has been found at autopsies. The giants themselves often have enormous hands and feet; they are feeble and short lived. In some instances kyphosis has developed later in life, with more pronounced symptoms of acromegaly.

Hyperostosis Cranii.—This is an enlargement of the cranium, beginning in early life, involving the anterior part of the skull rather more than the posterior, and leading to enormous thickening of the bones, from 1 to 3 or more cm. (0.5 to 1.5 in.). Other bones are sometimes affected. The symptoms sometimes resemble those of cerebral tumor, the cranial nerves being affected by the bony growth. Headache is common, and optic neuritis occasionally occurs. Marked exophthalmus is sometimes produced by the growth of the orbital bones which fills up the orbital cavities. The trouble is supposed to be of trophic origin, although the changes in the bones resemble those of inflammation. The involvement of the cranial vault and the absence of kyphosis and changes in the extremities render it easily distinguishable from acromegaly. Treatment has thus far proved unavailing.—K.]

Scleroderma.—In connection with acromegaly, we wish to refer briefly to scleroderma, which in a way is the opposite of acromegaly, and is perhaps also associated with the function of the hypophysis. In scleroderma we are not dealing with a hyperplasia, but with an atrophy and shrinking of the bones and soft tissues. The disease affects the hands and arms primarily. The fingers grow small, the terminal phalanges atrophy more and more, and the skin becomes smooth, glossy, and atrophic. The muscles and fasciæ are affected by the atrophic process. [In very rare cases there may be atrophy of one half of the body.—K.]

In the face the atrophy affects, above all, the lips and nose, and then the ears. The skin of the trunk and the soft parts of the thorax may also become involved, so that, as I observed in one case, distressing dyspnœa, in consequence of the interference with the respiratory movements, may arise. Not infrequently the atrophic stage is preceded by a stage of swelling. Occasionally the process is not diffuse at first, but appears in solitary disseminated foci. The disease occurs with markedly greater frequency in women than in men.

The prognosis is entirely unfavorable, but some symptomatic improvement may be obtained by massage, medical gymnastics, ointments, baths, etc. [Some cases apparently recover.—K.] Injections of thiosinamin have also been recommended. The administration of pituitary gland has given some interesting results.

Progressive Facial Hemiatrophy (*unilateral progressive facial atrophy*).—Unilateral facial atrophy is an extremely rare disease, which consists of a very slow and gradual, but usually constantly progressive, atrophy of one half of the face, affecting the skin, and also the fatty tissue, the muscles, and the bones, either in a uniform or a very diverse manner. The affection usually begins in youth. The female sex seems to be more disposed to the disease than the male.

The atrophy, which has its seat much more frequently on the left side than on the right, begins usually in a circumscribed spot either on the cheeks or on the chin. The skin, as a rule, gradually assumes a whitish or brownish color. The affected part, and finally the whole half of the face, gradually sink in more and more, so that the disease can be recognized at the first glance. The atrophy shows a sharp limitation at the median line. In many cases the muscles apparently remain almost wholly intact, but in some cases they show a marked atrophy, especially the muscles of mastication. The corresponding half of the tongue and the soft palate has sometimes been found implicated. Exceptionally, the atrophy involves the neighboring region of the shoulder and the upper extremity. The bones also atrophy, especially in the cases which arise in early youth. The hair on the affected half of the head often falls out in great amount, and becomes thin and atrophic. The sensibility remains perfectly intact. Marked vasomotor and secretory disturbances have only rarely been observed. The accompanying illustration (Fig. 213) shows a patient who was described by Romberg many years ago, and who for a long time frequented the German clinics in order to show himself.

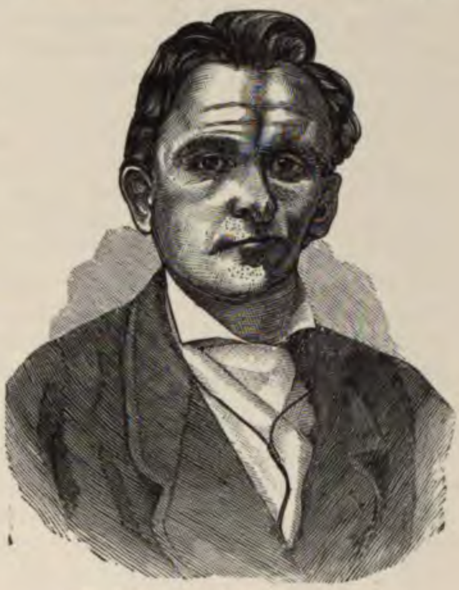

FIG. 213.—Left facial hemiatrophy.

Nothing definite is known in regard to the nature of the affection. Most observers at present agree that it is a trophic neurosis, an affection of trophic nerves or nerve centers, but where we are to look for the special seat of the disease, whether in the trigeminus or in the sympathetic, we do not know. Mendel lately found, in a case which came to autopsy, a definite neuritis in the trigeminus.

The disease is not dangerous in itself, and usually causes no special subjective disturbance, but it seems tò be incurable. In cases at their beginning we can at most make an attempt to bring the disease to a standstill by a long-continued application of electricity.

As an appendix it may be briefly mentioned here that there is a unilateral hypertrophy, which is also possibly connected with neurotrophic disturbances. We saw at Leipsic a ten-year-old boy, otherwise perfectly healthy, in whom a striking hypertrophy of the left side of his face and of the left arm had gradually developed.

Hydrops Articulorum Intermittens.—This is the name of a very rare disease, in which large swellings, usually of the knee joint, but sometimes of the other large joints, develop at perfectly regular intervals of one to four weeks. These swellings of the joints run their course without fever, and usually without any great pain, and disappear again in a few days. Such attacks may be repeated at intervals of different lengths during many years. Their nervous character is attested especially by the rapid onset and disappearance of the affection, and also by the combination of it with other nervous disturbances, such as angina pectoris, exophthalmic goiter, vasomotor symptoms, hysterical symptoms, etc., which combination has often been observed. In regard to treatment, salicylic acid, quinin, Fowler's solution, and subcutaneous injections of ergotin have been tried, but the trouble is usually very obstinate.

Disturbances of Secretion.—In addition to the vasomotor and trophic disturbances we must consider the disturbances of secretion. These are not infrequent. We have already learned to recognize anomalies in the secretion of saliva in facial paralysis, and of the lachrymal secretion in trigeminal neuralgia. Analogous symptoms are occasionally noticed in other nervous diseases. Disturbances of the sweat secretion are the easiest to confirm. Our understanding of them comes substantially from the discovery of the "sweat nerves," arising mainly from the sympathetic, which was made by Luchsinger. In nervous patients we have seen quite frequently, on the one hand, an abnormal increase of the sweat secretion (*hyperidrosis, ephidrosis*), and, on the other, a diminution or a complete disappearance of it. The former is seen on the paralyzed side in many hemiplegias and in spinal paralyses, the latter in tabes dorsalis. Anomalies of the sweat secretion are quite frequent, and are usually combined with vasomotor disturbances in certain general neuroses, such as hysteria and neurasthenia. In a few rare cases a genuine *hematidrosis* (bloody sweat) has been confirmed. [Colored sweating (*chromatidrosis*) has been reported in some very rare cases of hysteria.—K.] The condition known as unilateral hyperidrosis (unilateral sweating) is also especially interesting. In this there is an abnormal secretion of sweat, chiefly in one half of the face, more rarely in one arm or over the whole of one side. The affection has usually been observed in connection with tabes, syringomyelia, hemicrania, exophthalmic goiter, hysteria, etc., and, in at least a number of cases, it seems to be due to direct lesions of the sympathetic. On the other hand, we have repeatedly seen persons, who were otherwise perfectly healthy, in whom the secretion of sweat, coming on under normal conditions from heat or physical exertion, remained limited to one half of the body, especially the face.

Injuries and Diseases of the Cervical Sympathetic.—In conclusion, we would briefly mention here the symptoms which have been observed in direct injuries of the cervical sympathetic, trauma, pressure of neighboring tumors, etc. If we have to do with a paralysis of the sympathetic, we see almost constantly on the affected side a contraction of the pupil from paralysis of the dilator pupillæ supplied from the sympathetic, in many cases associated with a slow reaction to light. We also frequently see a narrowing of the opening of the lids from paralysis of Müller's muscle, and in old cases a retraction of the bulbus oculi, flattening of the affected half of the face, and occasionally increased redness and warmth in the ear and cheeks from vasomotor disturbance. In a few cases we see an increased sweat secretion. We may add that, according to Möbius, the normal reflex dilatation of the pupil, from painful irritation of the skin of the face, is absent in paralysis of the sympathetic. The opposite symptoms are found in conditions of irritation of the sympathetic. The occurrence of sympathetic symptoms in certain injuries of the brachial plexus has already been mentioned above (page 244).

CHAPTER II

MIGRAINE

(Hemicrania. Megrim. Sick Headache)

Ætiology.—By migraine or hemicrania we mean a peculiar form of headache, coming on in separate attacks, usually unilateral, and associated with considerable disturbance of the general condition and with pronounced nervous gastric symptoms (complete loss of appetite, nausea, vomiting). The affection occurs in women and men, somewhat more rarely in the latter, and almost always begins in youth, generally at the period of puberty, although typical cases of migraine have been repeatedly observed in school children. Quite frequently, but not always by any means, the disease affects persons who must be regarded as "generally nervous," who are anæmic, or who suffer from disturbances of menstruation. Heredity often plays a part, since hemicrania is both hereditary as such, and often appears in families which have suffered from other nervous diseases, such as epilepsy, hysteria, or the psychoses. We may mention as exciting causes, which may be made answerable both for the onset of the disease and especially for the individual attacks, physical and mental overexertion, great mental excitement, disturbances of digestion, indulgence in alcohol, etc. [Eye strain (page 188) may rarely have some influence in causing migraine as well as other forms of headache, and a careful search for any errors of refraction should therefore be made.—K.] The special cause of the disease, however, is probably, as a rule, a congenital predisposition.

Many writers lay great stress upon certain vasomotor symptoms which sometimes accompany migraine, and therefore believe that the disease is to be considered in the main an affection of the sympathetic; but we must agree with Möbius that this assumption is very improbable, and that the accompanying sympathetic symptoms are probably only secondary and of reflex origin, in consequence of the pain, or are merely a part of the whole attack of migraine. We are still utterly ignorant of the special nature of migraine —that is, of the kind or the place of the change which forms the basis of the clinical symptoms. The conjecture is interesting, but of course wholly unconfirmed, that there is in the attacks an "auto-intoxication" of the body—that is, the action of some poison developing from time to time in the body itself—but we do not know upon what part of the nervous system this poison may act; we can merely conjecture that the place of irritation, upon which the pain and other migrainous symptoms depend, is in the brain itself (in the cortex or deeper parts). This conjecture is supported especially by the nature of many of the symptoms which accompany the pain of migraine—the sensitiveness of the sensory organs, the slight mental change, the scintillating scotoma, etc.

Symptomatology.—Migraine always comes on, as has been said, in separate attacks, which are repeated at intervals of varying lengths, although some cases often show a remarkably great regularity. The onset of the attack in women often has some relation to the menses; in other cases it is due to one of the exciting causes mentioned above.

The attack of migraine usually begins with certain prodromal symptoms, which the patient soon recognizes as sure signs of his approaching suffering. These prodromal symptoms consist of general uneasiness, discomfort, pressure in the head, vertigo, at times tinnitus, spots before the eyes, darkening of the visual field, chills, malaise, abnormal yawning, etc. In a short time the pain begins. It is felt most either in the anterior frontal region or in the temporal or parietal region; it generally shows a continuous character, not intermittent as in neuralgia, and it may increase to a very great intensity. Usually one side of the head (the left most commonly) is the chief seat of the pain, particularly the forehead and the neighborhood of the eyes; but it may also happen that the pain affects the right and left sides alternately, and it is sometimes not limited to one side, but embraces nearly the whole head. The scalp is usually hyperæsthetic on the affected side, and the points of exit of the nerves may be sensitive to pressure, but firm pressure on the head (with the hands or by tying a towel tight around it) sometimes gives relief.

The second chief symptom in migraine, besides the headache, is the complete loss of appetite and great nausea. Sometimes vomiting comes on only at the end of an attack, but often there is almost uncontrollable vomiting throughout the whole attack. The patient not infrequently notices that the vomitus has a very sour taste, and we have ourselves found a decided hypersecretion of gastric juice in several cases of migraine. The patients' general condition is almost always very much disturbed. They feel very wretched and dull, and are very sensitive to external impressions, any bright light, any noise, etc. They are incapable of any mental exertion. In many cases remarkable ocular symptoms occur (ophthalmic migraine)—slight or marked ptosis of one eyelid, bright flashes before the eyes, scintillating scotoma, and not infrequently pronounced hemianopsia. These symptoms very often forebode the special attack of pain. Other severe nervous symptoms, such as paræsthesia in the hands and fingers, tinnitus, disturbances of speech, etc., have occasionally been observed.

The vasomotor symptoms accompanying the headache may occur in two different forms. From them migraine was formerly divided into two subdivisions—*hemicrania sympathico-tonica* or *spastica*, and *hemicrania sympathico-paralytica* or *angio-paralytica*. In hemicrania spastica, first described by Du Bois-Reymond from observations on himself, the forehead and ear on the affected side are pale, the skin is cool, the temporal arteries contracted, the pupil is often decidedly dilated, the secretion of saliva increased; in short, there are a whole series of symptoms present which all agree in pointing to a condition of irritation of the sympathetic (*vide supra*). In hemicrania paralytica, however, which was first described by Möllendorff, also from observations on himself, the face is reddened on the affected side, it feels hot, the temporal arteries are dilated and pulsate strongly, there is sometimes unilateral sweating of the face, the pupil is contracted—all symptoms, therefore, which can depend only on a paralysis of the sympathetic.

As has already been said, the significance of all these symptoms is not absolutely certain. We must also add that this division is more theoretical than real, and that the cases which occur in practice cannot by any means always be inserted into one or the other typical scheme without further ceremony. The vascular symptoms are often only slight, conditions of paralysis and of irritation of the sympathetic sometimes seem to alternate with each other in the same attack, and we may even frequently meet with apparently contradictory symptoms at the same time, such as pallor joined with contraction of the pupil. The vascular changes, when present, are almost never unilateral, but they are approximately alike in the two halves of the face. In severe attacks the whole face usually looks pale and sunken, but the initial pallor may give place to redness.

The duration of the attacks of migraine differs very much. It usually lasts several hours or a whole day; then the pain gradually disappears, and there is often considerable vomiting and sometimes a profuse discharge of urine toward the end of the attack. In the intervals between the different attacks most patients are perfectly well and free from pain. There are also severe forms of migraine where the individual attacks may last for several days or more ("*état de mal*"). Without an accurate history these severe conditions of intense headache and persistent vomiting may readily be falsely interpreted.

The whole course of migraine is very chronic, and it may last for years and years. It is usually a trouble to which the patient finally becomes accustomed. We must generally be quite guarded in our prognosis, for many cases resist very obstinately all attempts at cure. We can give the patient only the consolation that the trouble generally disappears of itself in advanced life. It is not usually attended with any special danger. In only a few cases has it been noticed that attacks of hemicrania of years' duration have preceded a severe cerebral disease that developed later.

Diagnosis.—The diagnosis of migraine is not difficult, if we hold strictly to the definition of the disease. The laity, especially women, of course, delight in calling every headache and all sorts of other nervous and hysterical conditions "migraine." Especially characteristic of true migraine are the heredity, the beginning in youth, the occurrence in attacks, the accompanying vomiting, and the other attendant nervous symptoms. Errors in diagnosis may arise from the fact that attacks of symptomatic migraine may also occur in severe organic diseases (tabes, brain tumor, etc.). We have seen a patient who suffered for years with severe "migraine." He finally died, and the autopsy revealed a cysticercus in the fourth ventricle with secondary hydrocephalus.

Treatment.—Very many patients who suffer from migraine finally renounce any special treatment after they have exhausted all possible remedies. They withdraw to their rooms when the attack comes on, darken the windows, take nothing but some tea, Seltzer water, or cracked ice, put a cold compress about the head, perhaps try a foot bath, and for the rest wait quietly until the attack is over. In fact, our remedies for cutting the attack short are quite uncertain. They sometimes aid, but they often leave us in the lurch, especially if used repeatedly. We must note especially that narcotics, such as morphin, are almost always ill borne in migraine, and do no good; but antipyrin, sodium salicylate (30 to 45 gr. [gm. 2 to 3], in strong black coffee), aspirin, phenacetin, salipyrin, etc., in many cases undoubtedly have a favorable action. Which remedy acts the best must usually be tested in the individual case. We have ourselves seen excellent results formerly from sodic salicylate, and lately from antipyrin, pure or mixed with the other remedies mentioned; the attacks of migraine, when the remedy was taken immediately upon the onset of the first symptoms, have become distinctly milder, and have run their course more rapidly. Of course the action often ceases with time, and we must then try another of the remedies mentioned. Of the other remedies used we may mention guarana (*Paullinia sorbilis*), 0.5 to 1 drachm (gm. 2 to 4) of the powder, and caffein, or sodio-salicylate of caffein, in 3- to 5-gr. doses (gm. 0.2 to 0.3), which is sometimes very serviceable. Caffein is also the chief constituent of "migrainin," which has lately been much praised. On theoretical grounds, inhalations of nitrite of amyl, 3 to 5 drops on a napkin, have been used in spastic hemicrania, and subcutaneous injections of ergotin in the paralytic form (aqueous extract of ergot, 2.5; dilute alcohol and glycerin, each 5; or 1 part of dialyzed ergotin in 4 parts of distilled water—injections of either, ♏ iij to xv). The practical benefit, however, is very doubtful. Many other nervines, such as bromid of potassium and Fowler's solution, have been recommended for continued use, and also extract of cannabis indica; and, lately, nitrite of sodium, 2 parts in 120 of water, a teaspoonful one to three times a day, and nitroglycerin, in troches containing $\frac{1}{120}$ to $\frac{1}{60}$ gr. (gm. 0.0005 to 0.001). Their action is similar to that of nitrite of amyl. In ophthalmic migraine the treatment by large doses of bromid of potassium is praised, especially in France, as being very successful.

In many cases the general treatment is very important. Preparations of iron, sea bathing, a mountain residence, and cold-water cures are often of decided service. We have seen remarkably good permanent results in several cases of severe migraine from the moderate use of Carlsbad water, either at Carlsbad itself or taken at home. We always strongly recommend a trial of this, especially when there are also symptoms of hypersecretion of the gastric juice (*vide supra*). With the moderate use of Carlsbad water we usually combine methodical hydrotherapy (cold sponging, half baths with douches, etc.) and an appropriate diet. The persistent application of electricity has also shown some good results, but we must not build very great hopes upon it. In the spastic form the action of the anode on the sympathetic is especially recommended, and in the paralytic form the action of the cathode, while the other electrode is to be placed on the cervical cord, or as high as possible on the occiput in the region of the medulla. Cautious galvanization of the head, and weak primary faradic currents, may also be used. Specialists in massage praise their mode of treatment in migraine; they massage either certain painful spots in the head or the gastric region. Finally, we must mention that migraine often seems to be connected with diseases of the nose, especially with hyperplasia of the erectile tissue in the nose, and that in such cases treatment of the primary disease with the galvano-cautery may result in the cessation of the migraine.

CHAPTER III

EXOPHTHALMIC GOITER

(*Parry's Disease. Morbo di Flajani. Graves's Disease. Basedow's Disease*)

Ætiology.—The special group of symptoms to which the name of exophthalmic goiter has been given, and whose three cardinal symptoms are acceleration of the pulse, goiter, and exophthalmus, was first carefully described in Germany in the year 1840 by the Merseburg physician Basedow, although similar but less precise observations had been published in England by Graves five years earlier. [Parry, in England, was the first accurately to describe the disease in 1786, and it was also recognized by Flajani in Italy in 1800.—K.]

We will discuss first those ætiological factors which, according to general clinical experience, appear to have some influence on the origin of this disease, reserving until later the consideration of the true nature of this affection. All those factors must first be mentioned which are important in the ætiology of neuroses in general. In many cases an hereditary predisposition can certainly be discovered. The disease has been repeatedly seen in several members of the same family. We have ourselves seen severe exophthalmic goiter in two sisters. Furthermore, exophthalmic goiter is also quite frequent in those families in which there is an hereditary predisposition to neuroses in general—epilepsy, the psychoses, or hysteria. Among the exciting causes we must first mention great mental excitement—grief, terror, anger. Sometimes real injuries, as well as these "psychical injuries," seem to have an influence on the development of the disease; that is, severe general bodily concussion, such as a fall. Many authors have laid considerable weight on diseases of the female sexual organs, but the importance of this factor seems to us to be exaggerated. It is certain, however, that the first symptoms of exophthalmic goiter often develop at the period of pregnancy.

The influence of sex upon the origin of the disease is plain, since women, especially somewhat anæmic, "nervous" women, are much more frequently affected than men. Exophthalmic goiter usually appears in middle life, while it is seen only exceptionally in children and old people.

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Physical examination of the heart shows an accelerated and, above all, the peculiar increased, lively, and diffuse cardiac pulsation; but after prolonged duration of the disease we also find, as we can affirm from many cases in our own experience, a manifest hypertrophy of the left ventricle, and also dilatation of the heart, and even actual valvular disease. In the diagnosis of the latter some caution is necessary, because functional heart murmurs, especially at the base of the heart, are often present in exophthalmic goiter.

The goiter usually develops somewhat later than the first symptoms in the heart. In many cases it is entirely absent, or present only in a slight degree. The swelling of the thyroid gland is very marked in only a part of the cases. There are sometimes decided variations in it in the course of the same case. The comparative softness of the tumor, the frequent and strong pulsations in it, and the loud vascular murmurs, which are often but not always heard, and which arise in the dilated vessels of the thyroid gland, are characteristic of the goiter in Basedow's disease. By laying the hand on it we can often feel the thrill and pulsation.

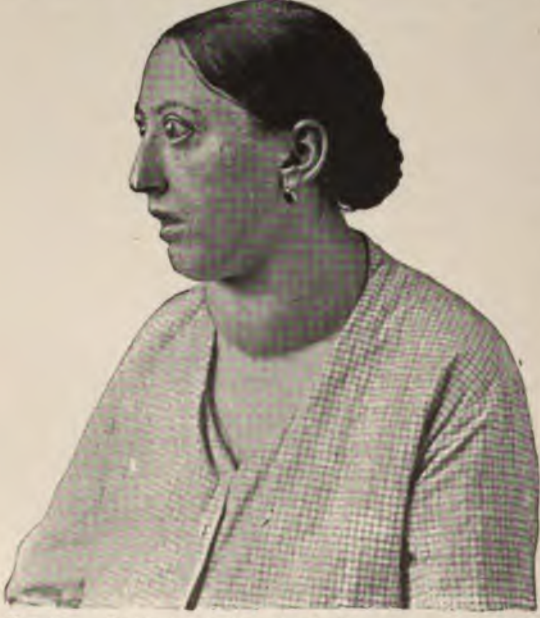

Fig. 214.—Patient with Basedow's disease (exophthalmic goiter). (Personal observation.)

The exophthalmus, the protrusion of the eyeballs from their orbits, is almost always bilateral, although it is sometimes more marked on one side than on the other (*vide* Fig. 214). We have seen marked unilateral exophthalmus only once in exophthalmic goiter. The cause of the exophthalmus is still obscure. Dilatation of the blood vessels at the back of the orbit crowding the bulb forward, has been suggested. A hypotonia of the ocular muscles is, however, more probable. In many cases it is entirely absent; in others it may attain so high a degree that an actual "dislocation of the eyeball" has been described. In the marked degrees of exophthalmus there is often a peculiar staring expression to the countenance. This is due chiefly to the unusual size of the palpebral fissure. The infrequency of winking, emphasized by Stellwag, has not been, as a rule, observed by me in my cases. On the other hand, a peculiar symptom, first described by von Graefe, is very important. On raising and, still more noticeably, on lowering the eyes, the corresponding associated movements of the upper eyelid, which are always present under normal conditions, are absent. This "Graefe symptom" may sometimes be one of the earliest signs of the disease, and may therefore be of diagnostic value. If present, it is very striking and characteristic. The best method of testing it is to have the patient fix his gaze upon an object held above his eyes. Then, with the head kept still and tilted slightly backward, the object is slowly moved downward and the patient is asked to follow it with his eyes. If Graefe's sign be present, the eyeball is lowered more than the upper lid, so that a portion of the white of the eye becomes plainly visible between the upper margin of the iris and the upper eyelid. In some cases of marked exophthalmus, the faulty lowering of the upper lid appears to be partly, at least, purely mechanical. However, Graefe's sign is sometimes observed in cases with slight or even no exophthalmus, and here it evidently indicates an interference with the synergic activity of the muscles. Disturbances of the pupils and of accommodation are unknown in exophthalmic goiter, but paralyses of the external ocular muscles sometimes occur, and we have ourselves repeatedly observed anomalies in the movements of the eyeball, especially temporary strabismus. We might mention one symptom, which Möbius first noticed, and which we also have repeatedly, but not constantly, seen, especially in patients with rather marked exophthalmus. It consists in the fact that one eye very soon deviates outward if we have the patient converge the eyes strongly, as in fixation of a near object—"insufficiency of convergence." Inflammatory processes in the eye are sometimes seen, which are probably due to the fact that the upper lid cannot so well protect the protruding eye.

Besides the chief symptoms of exophthalmic goiter thus far described, we must also mention a list of other symptoms which come under our observation, both in the typical cases and still more often in less pronounced cases—the so-called "*formes frustes*" of the French. Among them are some nervous symptoms, especially a peculiar rapid tremor to which Marie first called attention. This tremor affects the whole body or the extremities alone; it shows at times temporary remissions and exacerbations, and it may be so severe as to form the patient's chief complaint. A moderate tremor of the fingers and hands is one of the most constant symptoms of exophthalmic goiter. In a case under our observation marked tremor was one of the first symptoms of the disease. It may become so violent at times that there are even spasmodic twitchings in the extremities and in the muscles of the face. We may also mention headache, vertigo, weakness of memory, and sleeplessness. The peculiar nervous anxiety and the irritable disposition of the patient are, however, very frequent and in fact very characteristic, in many cases of the disease. The anxiety and the haste in all movements, in speaking, etc., often show themselves, even during the physician's examination, in so striking a way that they must be regarded as not unimportant factors in diagnosis. The easily occurring flushing of the face, the marked subjective feeling of heat, and the hot hands, from which many patients suffer, are probably due to vasomotor disturbances. Objective elevations of temperature up to 100° or 101.5° F. (38° to 38.8° C.) have also

Treatment.—Very many patients who suffer from migraine finally renounce any special treatment after they have exhausted all possible remedies. They withdraw to their rooms when the attack comes on, darken the windows, take nothing but some tea, Seltzer water, or cracked ice, put a cold compress about the head, perhaps try a foot bath, and for the rest wait quietly until the attack is over. In fact, our remedies for cutting the attack short are quite uncertain. They sometimes aid, but they often leave us in the lurch, especially if used repeatedly. We must note especially that narcotics, such as morphin, are almost always ill borne in migraine, and do no good; but antipyrin, sodium salicylate (30 to 45 gr. [gm. 2 to 3], in strong black coffee), aspirin, phenacetin, salipyrin, etc., in many cases undoubtedly have a favorable action. Which remedy acts the best must usually be tested in the individual case. We have ourselves seen excellent results formerly from sodic salicylate, and lately from antipyrin, pure or mixed with the other remedies mentioned; the attacks of migraine, when the remedy was taken immediately upon the onset of the first symptoms, have become distinctly milder, and have run their course more rapidly. Of course the action often ceases with time, and we must then try another of the remedies mentioned. Of the other remedies used we may mention guarana (*Paullinia sorbilis*), 0.5 to 1 drachm (gm. 2 to 4) of the powder, and caffein, or sodio-salicylate of caffein, in 3- to 5-gr. doses (gm. 0.2 to 0.3), which is sometimes very serviceable. Caffein is also the chief constituent of "migrainin," which has lately been much praised. On theoretical grounds, inhalations of nitrite of amyl, 3 to 5 drops on a napkin, have been used in spastic hemicrania, and subcutaneous injections of ergotin in the paralytic form (aqueous extract of ergot, 2.5; dilute alcohol and glycerin, each 5; or 1 part of dialyzed ergotin in 4 parts of distilled water—injections of either, ♏ iij to xv). The practical benefit, however, is very doubtful. Many other nervines, such as bromid of potassium and Fowler's solution, have been recommended for continued use, and also extract of cannabis indica; and, lately, nitrite of sodium, 2 parts in 120 of water, a teaspoonful one to three times a day, and nitroglycerin, in troches containing $\frac{1}{120}$ to $\frac{1}{60}$ gr. (gm. 0.0005 to 0.001). Their action is similar to that of nitrite of amyl. In ophthalmic migraine the treatment by large doses of bromid of potassium is praised, especially in France, as being very successful.

In many cases the general treatment is very important. Preparations of iron, sea bathing, a mountain residence, and cold-water cures are often of decided service. We have seen remarkably good permanent results in several cases of severe migraine from the moderate use of Carlsbad water, either at Carlsbad itself or taken at home. We always strongly recommend a trial of this, especially when there are also symptoms of hypersecretion of the gastric juice (*vide supra*). With the moderate use of Carlsbad water we usually combine methodical hydrotherapy (cold sponging, half baths with douches, etc.) and an appropriate diet. The persistent application of electricity has also shown some good results, but we must not build very great hopes upon it. In the spastic form the action of the anode on the sympathetic is especially recommended, and in the paralytic form the action of the cathode, while the other electrode is to be placed on the cervical cord, or as high as possible on the occiput in the region of the medulla. Cautious galvanization of the head, and weak primary faradic currents, may also be used. Specialists in massage praise their mode of treatment in migraine; they massage either certain painful spots in the head or the gastric region. Finally, we must mention that migraine often seems to be connected with diseases of the nose, especially with hyperplasia of the erectile tissue in the nose, and that in such cases treatment of the primary disease with the galvano-cautery may result in the cessation of the migraine.

CHAPTER III

EXOPHTHALMIC GOITER

(*Parry's Disease. Morbo di Flajani. Graves's Disease. Basedow's Disease*)

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We will discuss first those ætiological factors which, according to general clinical experience, appear to have some influence on the origin of this disease, reserving until later the consideration of the true nature of this affection. All those factors must first be mentioned which are important in the ætiology of neuroses in general. In many cases an hereditary predisposition can certainly be discovered. The disease has been repeatedly seen in several members of the same family. We have ourselves seen severe exophthalmic goiter in two sisters. Furthermore, exophthalmic goiter is also quite frequent in those families in which there is an hereditary predisposition to neuroses in general—epilepsy, the psychoses, or hysteria. Among the exciting causes we must first mention great mental excitement—grief, terror, anger. Sometimes real injuries, as well as these "psychical injuries," seem to have an influence on the development of the disease; that is, severe general bodily concussion, such as a fall. Many authors have laid considerable weight on diseases of the female sexual organs, but the importance of this factor seems to us to be exaggerated. It is certain, however, that the first symptoms of exophthalmic goiter often develop at the period of pregnancy.

The influence of sex upon the origin of the disease is plain, since women, especially somewhat anæmic, "nervous" women, are much more frequently affected than men. Exophthalmic goiter usually appears in middle life, while it is seen only exceptionally in children and old people.

Symptomatology.—The increased frequency of the pulse and the morbid excitability of the heart are the most constant and earliest symptoms of exophthalmic goiter. The frequency of the pulse averages from 100 to 120, rarely less, but in some cases even 140 or 160. It is not alike at all times, but has many variations, lasting for long periods and also coming on in single paroxysms. In sleep the frequency of the pulse, as we have ourselves confirmed, is sometimes almost normal, and sometimes rapid, but usually less so than when the patient is awake. A very vigorous action of the heart no means rare. In some acute and apparently very severe cases especially we have seen almost complete recovery; there was usually only a certain amount of exophthalmus after all the other symptoms had disappeared. The disease sometimes terminates fatally with signs of general weakness, and finally paralysis of the heart, but more frequently from complications in the heart or lungs.

In fully developed cases of exophthalmic goiter a positive diagnosis can almost always be made without difficulty. The peculiar facial expression, altered by emaciation and exophthalmus and associated with goiter, often permits the recognition of the disease at the first glance; but the diagnosis is often very uncertain in the milder, undeveloped cases. In many patients with slight or even marked goiter we find some "Basedow symptoms" without being able to call it actually "Basedow's disease." If there be no evident goiter, we must be very slow in our judgment, but absence of exophthalmus with other plainly developed symptoms is often seen. On the other hand, when there really is exophthalmus it is one of the most unequivocal symptoms. In all doubtful cases we should consider carefully the mode of onset and the associated symptoms, especially emaciation, tremor, general nervous irritability, the feeling of heat, the tendency to sweating, etc.

We have still to describe briefly those cases in which the cardiac symptoms (palpitation, dyspnœa, at times associated with marked cardiac hypertrophy or dilatation) are particularly pronounced, and for which no other cause can be made responsible than a concomitant struma. Cases of this sort are often described at present as "strumous heart." In our opinion, however, no essential distinction can be maintained between "strumous heart" and exophthalmic goiter. On careful examination some other symptoms of exophthalmic goiter (emaciation, tremor, general nervous irritability, etc.) can generally be found in cases of strumous heart. In all these cases we are consequently dealing with that large group of morbid symptoms which are due to a disturbed function of the thyroid gland.

Treatment.—In the first place we must consider the general treatment of the patient. For all severe cases of exophthalmic goiter it is my custom, in the first place, to prescribe a rest cure for several weeks. An open-air rest cure on a veranda, or in a garden or similar place, is the best. The more favorable the climatic conditions are, the better for the patient (high altitudes, southern climates, seashore). According to their condition, the patients must lie down the whole or, at least, the major part of the day. Careful alimentation should be combined with this (not too much meat, more carbohydrates, fats, milk, vegetables, prepared foods, etc.) in order to increase the weight of the body, if possible. Furthermore, baths are very much to be recommended—above all, carbonic-acid baths, or, possibly, salt and electric baths. Simple half baths, friction with cold water or brandy, vinegar, etc., are also often of use. Internal remedies may be divided into the general tonics (iron, arsenic) and the "specifics," which have been produced and recommended in view of the theory of the thyroid origin of exophthalmic goiter. Möbius himself recommended an "antithyroidin serum," prepared from serum of thyroidectomized sheep. The daily dose of this is ♏ xv to lxxv (1 to 5 c.c.). There is a powder, "rodagen," on the market, which is prepared from the milk of thyroidectomized goats. Many observers claim to have obtained good results with these preparations. I have also used them in many cases, but have never been convinced of any particular specific action. [Favorable results have also been reported from the use of a special serum proposed by Rogers and Beebe.—K.] The administration of thyroid tablets is probably in most cases distinctly injurious, but good results have been also claimed for it in a few cases! Personally, I believe that I obtained good results in a few cases by the administration of small doses of sodium iodid. But care must always be exercised in the administration of the iodid.

Many surgeons have great confidence in the operative treatment of exophthalmic goiter. If the disease depends on an abnormally increased and morbid activity of the thyroid gland, then the partial removal of this organ, or the ligation of its arteries, could certainly be of service. In fact, Kocher and other operators report numerous brilliant results. I, myself, have seen a few good results, though in many cases, it must be confessed, no marked benefit could be seen from the operation. The operation is not without danger; I have seen many fatalities from it. As a rule, therefore, one should first attempt to benefit the disease in other ways. If nothing helps, and if the case is a severe one, we are justified in recommending an operation. The attempt has also been made to influence the goiter, and consequently the whole disease, by means of Röntgen rays. In our clinic distinctly favorable results have not yet been obtained. Nevertheless, the therapeutic attempts with Röntgen rays deserve to be continued.

Of other remedies, electricity is first to be mentioned, especially the application of galvanism to the neck—the so-called galvanization of the sympathetic at the inner border of the sternomastoid. The slowing of the pulse, which sometimes comes on at once ("vagus irritation," or mental quieting!), is striking. Vigouroux praises faradization of the sympathetic and of the goiter as the best method of treatment. Among internal remedies we may recommend atropin or tincture of belladonna, sodium phosphate in daily doses of ʒj to ʒijss. (gm. 5 to 10), and ergot or ergotin. We believe we have repeatedly seen good results from the latter. The phosphorus-albuminous preparations (phytin, protylin, etc.) are occasionally of use. Concerning the administration of iodin, sufficient has already been said. Digitalis has often been prescribed for the palpitation, but usually without any good result. In many cases, however, with threatened cardiac weakness, repeated trials of digitalis, strophanthus, caffein, valerian preparations, etc., will nevertheless have to be made. With great exophthalmus the eyes must be protected from external injuries. The occasional severe symptoms on the part of the digestive organs (vomiting, diarrhea) must be treated symptomatically, by ice, opium, lupulin, and champagne.

CHAPTER IV

EPILEPSY

(Falling Sickness. Morbus sacer)

Ætiology.—Epilepsy is a peculiar disease of frequent occurrence, the main symptom of which is paroxysmal loss of consciousness. In typical cases the unconsciousness is associated with violent general convulsions, but there are many anomalous and rudimentary forms of epilepsy without any symptoms of motor irritation. "Genuine epilepsy" is a functional neurosis—that is, with our present means of investigation we can discover no constant objective lesion of the nervous system as its basis. As many of the patients feel perfectly well for weeks and months in the interval between the attacks, there can be no question of any marked permanent anatomical changes, at least in the beginning. It is true, however, that in epilepsy of long standing, marked and persistent mental disturbances (*vide infra*) frequently occur, so that a permanent anatomical change in the brain may be presupposed, and is also generally demonstrable in the advanced cases. The symptomatic epileptic attacks must be distinguished from "true epilepsy." Attacks entirely similar to those of true epilepsy not infrequently occur in the course of tumor, syphilis, and other diseases which do present an anatomical lesion; but such attacks are merely symptomatic, and are therefore termed "epileptiform," in distinction from the genuine epileptic paroxysms.

Of the actual causes of epilepsy we know scarcely anything. We are acquainted only with certain factors which are favorable to the development of the disease, and are to be regarded, therefore, as predisposing or exciting causes. Heredity is decidedly the most important of these. About one third of all cases of epilepsy occur in persons who have inherited a nervous diathesis, and one or more of whose blood relations have suffered from disease of the nervous system. It should not be understood that we must find other cases of genuine epilepsy in the family, in order to establish the fact of congenital predisposition. The question is merely whether the ancestors have exhibited a general tendency to nervous disease. The more accurate and careful our investigations, the oftener do we find among the relatives of the patient instances of nervous trouble—sometimes genuine epilepsy, sometimes insanity, hysteria, or general "nervousness." Of course these "nervous families" present, besides those that are actually ill, others who are more or less peculiar and odd, and yet others gifted with extraordinary talents, although frequently somewhat ill-balanced. It is said that the children of parents who are related to each other are somewhat predisposed to epilepsy, as well as to other nervous diseases, but certainly this factor is very rarely of importance. It is only of importance if both parents are descendants of the same family with hereditary nervous history. Perhaps drunkenness in the parents is somewhat more prejudicial in this regard. It is said to have been repeatedly observed that children begotten while the father was intoxicated became epileptic.

[Statistics vary greatly as to the percentage of neuropathic heredity in epilepsy (eleven to eighty-seven per cent) and as to the percentage of epilepsy itself (similar heredity) in the antecedents of the epileptic (one to thirty-seven per cent). It must not be forgotten that there is a very considerable percentage (sixty per cent or more) of nervous affections to be found in the antecedents of healthy people, very nearly as great as can be found in the antecedents of the neuropathic and insane. It is far more important in all these affections to discover any noxious agents in the antecedents which may injure the germ plasma. The germ plasma may be injured by various causes, such as poisons (alcohol, morphin, lead), infection (syphilis, tuberculosis, or acute disease at the time of procreation and gestation), constitutional disease (anæmia, leukæmia, gout, chlorosis, diabetes, arthritis deformans), or local diseases of the generative tract. Such factors existing at the time of procreation or during the period of pregnancy will tend to produce offspring that are in some way defective, in whom various forms of nervous or mental disease can readily develop.—K.]

There are other influences which are assumed to have ætiological importance, but whether justly or not is difficult to decide. Alcoholic excesses certainly act in this way only in isolated cases (although epilepsy is said to attack absinthe drinkers in France quite frequently). Venereal excesses probably have still less importance. It should also be borne in mind that not infrequently excesses in these directions are the result of neurotic tendencies already existing. Syphilis has no direct connection with genuine epilepsy. Epileptiform convulsions may, as we have seen previously, be symptomatic of syphilis, being due to the cerebral lesion caused by this latter disease. It is possible that hereditary syphilis plays a rôle in some cases of apparently genuine epilepsy, but it has not been proved. Certain factors may determine the onset of epilepsy, although they cannot be said to cause the disease (except the symptomatic epilepsy which develops after a previous encephalitis). Such are overexertion of mind or body, repeated emotional disturbance, certain general conditions of the system, such as anæmia or malnutrition on the one hand, and plethora on the other; and, in particular, acute febrile diseases, such as scarlet fever, measles, and gastric diseases. [The individual attack is often brought on by some disturbance of digestion or indiscretion in diet.—K.] Another important point is that the first attack is sometimes brought on by great mental excitement, especially fright. But here, too, it is probable that the terror is merely the exciting cause, a tendency to the disease already preexisting. We must also be on our guard against confounding genuine epilepsy with the convulsive form of hysteria (*q. v.*), which very frequently develops after fright.

In some instances there is an evident connection between epilepsy and a previous injury to the skull at birth or from a fall, or blow, etc. At a certain interval after the trauma, attacks begin which seem precisely like those of genuine epilepsy. This is known as "traumatic epilepsy." These are not cases of genuine epilepsy, however, inasmuch as there is really some anatomical lesion of the cortex cerebri which, in some way as yet unknown, causes irritation of the motor centers of the cortex (*vide infra*). It is often the case that this variety of epileptiform attacks is peculiar in that the convulsions are at first unilateral, or confined to a single limb, corresponding to the seat of the injury in the opposite cerebral hemisphere.

"Reflex epilepsy," finally, deserves special notice. This name is applied to cases where each convulsive attack seems to be excited by reflex influences originating in some remote part of the body. Most cases have followed injury of peripheral nerve trunks (retained splinters, or scars), and have ceased upon removal of the exciting cause. Other causes are new growths in the nerves, foreign bodies in the ear, otitis, intestinal parasites, and, apparently, diseases of the female sexual organs. It seems probable that sufferers from these attacks have had a tendency to disease of the nervous system. We must hesitate to rank reflex epilepsy in the same class with the genuine form. The whole subject of "reflex epilepsy" needs a new and thorough investigation. It has been in the past so often confounded with hysterical ("traumatic-hysterical") attacks that we are personally disposed to regard most reports of reflex epilepsy with extreme skepticism.

Both the traumatic and the reflex varieties of epilepsy have repeatedly been the object of experimental investigation. Brown-Séquard has shown, by a great number of experiments, that epilepsy can be excited in rabbits by injuries to the medulla, the spinal cord, and the sciatic, as well as other peripheral nerves. A certain time after the operation the animals undergo spontaneous convulsive paroxysms. These occur at frequent intervals and for a long time, and they may be voluntarily excited at any time by irritation of a certain portion of the skin called the "epileptogenous zone." An interesting observation in this connection has been made by Brown-Séquard, which is that sometimes the progeny of these animals, who have been made epileptic, suffer from spontaneous epilepsy. Westphal induced epilepsy in guinea pigs by blows upon the skull. Immediately after the blow general convulsions occurred, but soon entirely ceased. Afterwards, however, there were repeated epileptiform attacks. Westphal thought that the causative lesion in these instances was the minute hemorrhages which were found in the upper part of the cervical division of the cord and in the medulla.

These and other experiments, which will be spoken of later, interesting as they may be in themselves, have contributed absolutely nothing to our knowledge of genuine epilepsy. In regard to the latter we have nothing left but to assume that there is a congenital, abnormal predisposition of the brain, and especially of its motor centers, but the nature of this predisposition is still wholly unknown. Some recent theories of epilepsy will be briefly mentioned later.

Clinical History.—In describing the symptoms of epilepsy, we shall first consider the various forms of the epileptic paroxysm, and then describe the general course of the disease.

1. The fully developed epileptic paroxysm is usually described, for the sake of greater clearness, as made up of several stages. First is the prodromal stage, or, according to Galen's expression, still in vogue, the stage of the epileptic aura (*aura* = breath). Not infrequently, however, there is no aura whatever, the convulsions coming on without warning; but in many cases the prodromata are well marked, and are repeated with noticeable regularity and similarity before each individual attack, although the different cases of epilepsy differ greatly as to the special phenomena of the aura occurring in each.

The best manner in which to distinguish the various forms of aura is according to the nature of the nervous phenomena, whether sensory, motor, vasomotor, or psychical. Of these the most frequent is, beyond a doubt, the sensory. Here we have peculiar paræsthesiæ, beginning in the arm or leg, or perhaps in the region of the heart or stomach, and thence usually "mounting to the head." It is seldom that the peculiar sensation is actually like a "breath" or puff of air. The aura which proceeds from the epigastrium is sometimes associated with a very disagreeable feeling of oppression and anxiety, and often also with nausea and vomiting. The aura may be referred to the nerves of special sense. In certain instances the patient perceives an unpleasant odor, which he likens to some familiar one. An aura of taste also occurs, but it is very rare. An optical aura is much more frequent, consisting of a subjective sensation of color or light (usually a sensation of red appears first), of an apparent increase or diminution of the size of surrounding objects, or finally of actual hallucinations of vision, such as beholding all sorts of human or brute shapes. An auditory aura is not very rare; it produces a sudden feeling of deafness in one ear, or various subjective sounds, like whistling, humming, roaring, etc.

The motor aura takes the form of mild premonitory contractions, affecting the head, face, arm, or leg. There may be aphasic disturbances at the same time, or we may observe symptoms of irritation of the unstriped muscles (choking, or a desire to go to stool). Sometimes there are prodromal vasomotor phenomena, where the aura consists of a sensation of cold or warmth, often associated with excessive pallor or redness of the face or hands. An attack may be ushered in by chilliness, perspiration, or palpitation.

Finally, the name of psychical aura is applied to those initial symptoms which consist of vertigo, confusion of thought, or other pronounced disorders of consciousness. A particularly frequent form for this to assume is excessive mental uneasiness and excitement. We may also add that various forms of aura are not infrequently seen in combination.

The aura lasts sometimes only a few seconds. It may persist long enough for the patient, who knows from experience what is coming, to lie down or take other precautionary measures (*vide infra*). In some few cases the aura may last hours, and even days. This is especially true of the psychical variety. Sometimes the aura passes away without being succeeded by any true epileptic fit, but it is usually followed by the second stage of the attack—the convulsive stage.

The convulsive stage of the epileptic attack almost invariably begins abruptly. Perhaps there is no aura, or only a very brief one, before the patient falls suddenly to the ground, usually on his face, although sometimes on the side or back. Consciousness is entirely suspended. Insensibility is complete, and often the patient sustains severe injury from his fall. Some patients utter a loud "epileptic cry" at the commencement of the attack, when they are already entirely unconscious.

The convulsive attack begins with a brief period of general tonic spasm of the muscles. The head is usually strongly extended, the teeth are pressed firmly together, the trunk is curved backward in opisthotonos, the extremities are extended, and the fingers are clenched over the flexed thumb. Inasmuch as the respiratory muscles participate in the seizure, breathing stops, and the original pallor of the face soon gives place to deep cyanosis. This general tonic convulsion ordinarily is but brief, perhaps fifteen to thirty seconds. It is followed by the second period of the convulsive stage—that of the clonic convulsions. The facial muscles now exhibit the most violent contortions—the eyeballs twitch or present a conjugate deviation toward one side, the tongue is thrust out and retracted convulsively, the head beats violently against the floor, and the muscles of the arms, legs, and trunk undergo the severest clonic spasms. The pupils are probably contracted for a short time at first, but during the convulsive stage they are widely dilated, and do not react at all. The pulse is somewhat accelerated, but not greatly. The temperature is normal, or elevated a small fraction of a degree. The cutaneous reflexes are lost immediately after an attack; but the tendon reflexes are generally somewhat exaggerated, although sometimes they also are diminished or absent. Not infrequently an involuntary dejection takes place during the fit, or the bladder is emptied, and in men there may be a seminal emission. During these violent convulsions the body is often severely injured. The tongue is frequently bitten. The face, which is usually pale at the beginning of the attack, becomes extremely cyanotic. The veins in the neck are distended, and the venous stasis is so extreme that sometimes small hemorrhages occur into the conjunctiva, the skin of the face, and other parts.

The convulsive stage usually lasts only a few minutes. Then the contractions cease, often after a deep, long-drawn sigh, and the patient passes into the third stage of post-epileptic coma. He lies unconscious, but his respiration grows quiet, and the cyanosis vanishes. Gradually the coma yields to slumber, which may persist for some hours; but some patients remain only a very brief time in this stage, and recover from their attack with surprising rapidity. It is, however, not infrequently the case that for some days distinct sequelæ are felt; there are headache, languor, and exhaustion, and mental despondency and irritability. For some time there may be severe pain in the muscles, particularly those of the trunk. There may be slight transitory paresis of one limb or one side of the body after an attack. Immediately after the attack the Babinski toe reflex [the Oppenheim reflex and ankle clonus] can frequently be elicited. In the first urine passed after the seizure is often found a trace of albumen, and perhaps a few hyaline casts. Not infrequently there is also decided polyuria for some time subsequent to the fit. Almost always after recovery of consciousness there is no recollection of what has happened, or it is very imperfect. On careful investigation we often find that the memory of events immediately preceding the attack (for fifteen minutes to two hours before) has also entirely disappeared. A student, for example, who had an epileptic attack during my clinic, later had absolutely no knowledge of how he came to the clinic or of what he had seen or heard there before his attack. In a few cases (especially after traumatic epilepsy) this retrograde amnesia may extend over a much longer period.

2. *The Milder, Rudimentary Forms of Epileptic Seizure. Petit Mal.*—Besides these violent paroxysms just delineated ("*grand mal*"), there are very often witnessed in epilepsy milder attacks of so-called "*petit mal.*" Sometimes there is only a transitory dizziness, or slight faintness, or perhaps a brief loss of consciousness, but without accompanying symptoms of motor irritation. These milder attacks may or may not be preceded by an aura. Cases have been repeatedly seen where the patient suddenly pauses in the midst of conversation, card playing, piano playing, or other occupation, stares absently for a moment, and then, with equal abruptness, goes on with what he was doing, as if nothing had happened. In other instances the patient pursues his occupation during this brief suspension of consciousness. For example, if seized while upon the street, he walks on mechanically, but takes the wrong turning, or goes into a strange house, when suddenly he comes to himself and wonders to find himself where he is. Cases of "sudden somnolence" are also almost all of them ascribable to epilepsy. There are all sorts of transitional forms between the slight attack of vertigo and the typical epileptic fit. Not infrequently the patient falls down unconscious, but he has only a slight twitching

of the face or arm, and in a few minutes is entirely himself again. Very often, especially in children, the attack consists of a short, piercing cry, loss of consciousness, a few convulsive movements of the arms—and in a few seconds all is over. Such attacks come on in the daytime and often in the night as well. In other patients the attacks consist of sudden pallor, springing to the feet, rushing to the door, the ejaculation of some short words, etc. In short, it is hardly possible to describe all the different manifestations of the rudimentary and atypical epileptic attacks; but persons who suffer from such slight attacks not infrequently have perfectly typical and fully developed epileptic attacks besides, and this is of especial importance in diagnosis.

3. *Epileptoid Conditions* (*Epileptic Equivalents*).—Cases of *petit mal* are generally rudimentary forms of the typical epileptic paroxysm, and consist merely of a simple impairment of consciousness, possibly associated with slight motor symptoms; but in the epileptoid state the characteristic features of the typical epileptic attack are quite subordinate. The disturbance is paroxysmal, and it can often be shown to be connected with genuine epileptic seizures, else its undoubted relation to epilepsy would never have been recognized. The greatest practical importance attaches to the "psychical equivalents of epilepsy" (Samt). These are attacks of mental disturbance, which either immediately succeed a typical epileptic fit ("post-epileptic insanity") or occur independently. These attacks are manifested by states of complete mental confusion, in which the patient may do the strangest things—may strip himself, steal, jump into the water, or commit incendiarism. Sometimes these patients make long journeys when in this clouded condition, and are surprised after normal consciousness has returned to find themselves in entirely strange surroundings. [Most of these cases of *"fugue,"* however, are hysterical. —K.] Besides these "states of epileptic confusion" there are also attacks of violent psychical excitement, associated with terror, frightful hallucinations, and resultant maniacal excitement. Not infrequently the patient is led to acts of violence against those about him. In the young the attack may take a peculiar form—the child runs about in a peevish way, collects all sorts of things together, makes strange motions, etc. We have also frequently seen in children brief states of disturbed consciousness of sudden onset, with dominant ideas of apparent anxiety, often associated with some twitching. Many cases of so-called *pavor nocturnus* are certainly epilepsy, although it is sometimes not easy to exclude hysteria. Numerous and valuable particulars upon this subject, and a consideration of its great medico-legal importance, must be sought in text-books on insanity, as we cannot discuss them further here.

Among the rarer forms of epileptoid attack are the epileptoid sweatings (Emminghaus), a spontaneous outbreak of excessive perspiration in epileptics, which may or may not be associated with impairment of consciousness, and also attacks of epileptic tremor (Féré), procursive epilepsy (attacks when the patient runs impulsively forward), etc.

General Course of the Disease.—In a large majority of cases epilepsy begins before the thirtieth year. Often the disease appears in early youth, and sometimes even in the earliest years. Many a child has "convulsions from teething," which later on are seen to have been epileptic. It is only in rare instances that the first appearance of trouble occurs in advanced life. If either typical or rudimentary epileptic attacks appear for the first time late in life, in previously healthy people, we are generally not dealing with true epilepsy, but with a symptomatic attack caused by a totally different disease—particularly often, arteriosclerosis of the cerebral vessels; furthermore, occasionally, multiple sclerosis, brain tumors, contracted kidney, etc.

It is impossible to give any general rule as to the frequency of the paroxysms. Different cases differ very much. There are persons who during their whole life have no more than three or four seizures, at intervals of ten or fifteen years, while in most cases there is an attack every two to eight weeks. In other cases the fits may recur still more frequently, even daily. One very often sees certain variations in the course of the disease; at some periods the intervals between the attacks will be longer, and at others shorter. In severe cases the patient may have for several days very frequent seizures, so that he does not regain consciousness at all between them. This is termed the epileptic state (*état de mal, status epilepticus*). The condition is quite rare. It is often fatal, death being ushered in by a great rise of temperature.

External influences sometimes affect the frequency of epileptic attacks. Alcoholic or sexual excess, [indiscretions of diet], mental excitement, and physical overexertion almost always exert a malign influence. Alcoholic epilepsy proper has still to be considered separately—the epileptic attack occurs only after marked alcoholic excesses. Periodical dipsomania itself may even be an indication of an epileptic predisposition. In women the appearance of the catamenia is not infrequently the signal for the occurrence of an attack. In many instances the disease begins with the first establishment of menstruation. Sometimes, however, epileptic girls grow better when they arrive at puberty. Pregnancy sometimes increases and sometimes diminishes the frequency of the paroxysms. Intercurrent diseases, particularly acute infectious diseases, seem frequently to exert a beneficial influence upon the frequency of the attacks.

There is a practical distinction between diurnal and nocturnal epilepsy. Many patients have attacks only during the day, while others again have them only at night. A case of pure nocturnal epilepsy may go on for a long time unsuspected, particularly if the patient sleeps alone. He seldom has, the next morning, the slightest recollection of his attack during the night. He usually perceives, however, from a confused feeling in his head, or from certain injuries, such as a bitten tongue, or from the disordered state of the bed, that something must have happened to him during the night. In some cases of nocturnal epilepsy the patient wakes up out of sleep before he enters into the epileptic state of unconsciousness. Probably he is aroused by the aura. Besides cases where the fits occur during the day or the night only, mixed forms are frequently seen.

With regard to the occurrence of the different varieties of epileptic seizure, all sorts of combinations are possible. Many cases never have any but the typical convulsions, but in many others there are also a greater or less number of attacks of *petit mal*. The latter may even be for a long period the sole indication of the disease. Often there are no epileptoid conditions whatever, while in other instances the "psychical equivalents" are the most prominent feature of the disorder.

During the interval between the individual attacks many epileptics seem perfectly well, both physically and mentally. They are not infrequently, to be sure, somewhat peculiar and nervously excitable, or again dull and lethargic; but this does not by any means apply to them all. Many epileptics, and particularly such as have comparatively infrequent paroxysms, are very capable, and history furnishes numerous examples of eminent men who suffered from this disease—for instance, Cæsar, Mohammed, Rousseau, Napoleon I, and others.

Much effort has been devoted to the discovery of "signs of physical degeneracy" in epileptics. Relying upon numerous measurements, Benedikt believes that a majority of epileptics exhibit craniometric anomalies, such as asymmetry of the cranium, macrocephalia, or steepness of the vertex. It is also not unusual to meet with anomalies of the ears, teeth, or hands[1] in such cases. Indeed, all peculiarities of this sort are, in general, more frequently observed in neuropathic families than in healthy ones.

When the disease has lasted some time, and particularly if the attacks come at very short intervals, the general condition of the patient often undergoes a gradual but marked change. This rule is by no means invariable. The psychical disturbances, in particular, gradually predominate more and more. The character undergoes a peculiar change. The patients become irritable, and addicted to acts of violence; they show ethical defects, are inclined to lie, etc. In addition to this, the mental weakness becomes more and more pronounced. The intellect grows feeble, memory grows weaker, and occasionally there is finally a terminal dementia. In such cases the body also suffers. There are emaciation, paresis, tremor, and other persistent disturbances of cerebral origin. All these manifestations indicate that there is, nevertheless, a chronic degenerative process of the central nervous system at the basis of epilepsy, which, for a long time, is manifested almost entirely by the epileptic attacks, but finally leads to permanent disability.

In regard to the duration of epilepsy, it must be termed a life-long disease. To be sure, it is no rare thing for the paroxysms to cease and not return for years. But one can never rest satisfied that all trouble is at an end; some cause or other may excite another attack after a long interval. In general an epileptic has a shorter life-expectancy than healthy persons, especially as he may be carried off by chronic pulmonary or other intercurrent disease.

The prognosis is obvious from what has been already said. The individual seizure is only exceptionally dangerous of itself. The so-called "*status epilepticus*" often ends fatally, as above stated. In general those cases may be called the most favorable where the separate paroxysms are infrequent and mild, but even here the disease may suddenly assume an aggravated form. With regard to the distinction between nocturnal and diurnal epilepsy, the nocturnal is, in our opinion, the milder of the two.

Pathology.—The very fact that in the intervals between attacks the patient often betrays no sign of disease, shows that epilepsy cannot be due to any persistent macroscopic lesion of the tissues. Indeed, in many cases nothing is found at the autopsy, or, at most, changes which cannot be regarded as essential, such as osteosclerosis of the cranium or thickening of the cerebral meninges. Epileptic subjects who were during life decidedly demented usually present atrophy of the hemispheres. Meynert, and recently also some other investigators, state that changes in the pes hippocampi major are noticeably frequent in epilepsy; but these changes are not at all constant, and their significance remains to be established. Recent findings of changes in the cerebral cortex (neuroglia proliferation, etc.) in epileptics do not materially aid our understanding of the morbid symptoms. There is something seductive in the attempts which have been repeatedly made, to regard epilepsy as an auto-intoxication of the body. It is believed to have been demonstrated that the urine, blood, and also the cerebro-spinal fluid of patients suffering from epilepsy is more toxic for animals, especially immediately after the attacks, than are the same fluids from healthy or non-epileptic individuals. Certain chemical bodies (ammonium carbamate, cholin, etc.) have already been designated as the active toxic agents. However, all these assertions require further proof, and are partly equivocal in their interpretation, and partly entirely incorrect.

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[1] We have seen an epileptic who had six fingers on each hand.

We must therefore, for the present, be content to assume that the cause of the epileptic seizure is an intermittent functional condition of irritation. A natural question is: Where shall we locate this irritation, and what may be its nature? The opinion was long current that the medulla oblongata must be regarded as the true seat of the disease. Schröder van der Kolk was the first to express this opinion. It afterwards received support from the experimental investigations of Nothnagel, who demonstrated that irritation of a particular spot ("convulsive center") in the pons, in rabbits, invariably excites general convulsions. Nevertheless, most pathologists have now abandoned this view, because experiment and clinical observation indicate with increasing distinctness that the origin of epileptic convulsions is to be sought, at least in the great majority of cases, in the cerebral cortex. The clinical evidence is the invariable combination of convulsions and impairment of consciousness; the circumstance that the milder and the masked forms of epilepsy, now known to be intimately related to the true epileptic convulsions, also, almost without exception, indicate psychical disturbance; that attacks, the symptoms of which are perfectly analogous with those of epilepsy, are often found to be the result of anatomical lesions of the cerebral cortex; and, finally, that these convulsions in man and the convulsions of "cortical epilepsy" (*vide infra*), experimentally produced in animals, extend over the different groups of muscles in a way which corresponds precisely with the anatomical position of the different motor centers in the cortex (Hughlings Jackson). For example, if the convulsion begins in the distribution of the facial nerve, it extends from this point to the arm before it affects the leg.

There is also experimental evidence that epileptic paroxysms are of cortical origin. A great number of observers (Hitzig, Ferrier, Albertoni, Luciani, Franck and Pitres) have proved that electrical irritation of the motor regions of the cortex in animals will produce epileptiform convulsions. Unverricht has made the most thorough investigations in regard to this point upon dogs. He found that when a motor center is stimulated the convulsions spread from the corresponding group of muscles to others in a way which corresponds precisely to the anatomical position of the separate centers. If one of the centers in the cortex be destroyed, the convulsions of the corresponding muscles cease at once. This proves that the motor centers must be intact in order to render the occurrence of epileptic seizures possible. Just how the stimulation extends from one center to another we have as yet no certain information. Probably it travels horizontally through the cortex.

We see, therefore, that in all probability the seizures in man also originate in the cortex of the brain, although, of course, the involvement of the subcortical motor centers in the course of the attack cannot be excluded. The phenomena of the aura are likewise referable to some stimulation of the cortex, probably of the sensory region in most cases, as in the optical aura. The starting point of the irritation is usually in the motor cortex, but it does not seem impossible that an irritation developing in the posterior cortical regions may extend secondarily to the anterior motor portion of the cortex (Unverricht). The manner in which the irritation is created is as yet entirely conjectural. Kussmaul and Tenner proved that epileptiform convulsions can be excited by a general cerebral anæmia, and this fact was the foundation for the assumption that the genuine epileptic convulsions are also due to a temporary cerebral anæmia, caused, it may be, by local vasomotor constriction. Definite proof has not yet been furnished on this point. In the artificial epilepsy which Unverricht produced, and that also which Magnan excited by absinthe, the cortex of the brain was not strikingly anæmic. In regard to the occurrence of the attacks in true genuine epilepsy we can therefore provisionally make the imperfect assumption that, as the result of a diseased condition, the motor cortex of the brain has lost the power to store up its motor energy and to give it out again in normal fashion, and that therefore there arise, from time to time, morbid "discharges of energy" and an outbreak of the convulsive movements already described. We will omit a further discussion of this theory, since we would soon be lost in the realm of mere hypothesis.

Diagnosis.—Most cases of epilepsy are easily recognizable. It needs only to be borne in mind that epileptiform convulsions may also occur as a symptom of cerebral diseases which do have an anatomical basis, such as tumor, abscess, multiple sclerosis, and hydatids. As a rule, however, such diseases are readily distinguished by the state of the patient between the seizures, or by the further course of the illness. It should also be understood that unilateral convulsions, or such as are confined to a single member (Jacksonian epilepsy, *vide supra,* page 475), are usually, though of course not invariably, not true epilepsy, but symptoms of some circumscribed affection of the cortex. We will return again to the differentiation from hysterical convulsions in the chapter on hysteria. Weight should be laid on the general character of the attack, the complete loss of consciousness in epilepsy, the dilatation of the pupils, which do not react to light, the initial pallor not infrequently observed, and the subsequent cyanosis of the face. We may also consider the character of the onset and the duration of the attack (in true, genuine epileptic attacks the convulsive stage seldom lasts longer than a few minutes), the biting of the tongue and possibly other injuries arising from the fall at the beginning of the attack, the condition of the patient immediately after the attack (deep, continued sleep is in favor of epilepsy), the possibility of exciting the hysterical attacks voluntarily (by suggestion, by pressure on a "hysterogenous" zone, etc.), which we cannot do with epileptic attacks. Very frequently we have to make the diagnosis of epilepsy not from personal observation of an attack, but merely from the history furnished by the patient or his relatives. This is, as a rule, possible if we know exactly how to put the questions and for what we must inquire (nature of the attacks, their brief duration, possible injuries, etc.). As objective symptoms, aside from the sequelæ of any possible injuries, the general habitus (shape of the head, facial expression), and the general mental make-up of the patient, are not unimportant. In the detection of simulated epilepsy (where we generally have to do with hysteria), the same diagnostic features (pupils, manner of falling, condition of reflexes after the attack, etc.) must be considered.

Treatment.—Although no remedy is capable of effecting a sure and permanent cure of epilepsy, yet a favorable influence can be exerted upon the disease in many ways, so as to lessen the frequency and severity of the paroxysms, and to avert many of their evil results.

In the first place, regimen is of great importance. Any excessive exertion of mind or body must be forbidden. Temperance must be exercised in eating and drinking. Strong coffee and tea are permissible only in moderate quantities; it is best to prohibit alcohol; the patients also should not smoke too much. The diet should be simple and unirritating, and vegetable rather than animal. It is said that in some cases decided improvement has been brought about by confining the patient to milk and vegetables. In summer the patient should live quietly in the country or the mountains. We have also the individual constitution to consider—an abundant diet, or a restricted diet and aperient waters, etc., are prescribed, according to whether we are dealing with weak, anæmic individuals, or, on the other hand, with plethoric, corpulent persons. Mental exertion and excitement are to be avoided as much as possible. Physical exercise or medical gymnastics, gymnastics in general, etc., are possibly of advantage for robust individuals when used in moderation. I recall one epileptic in whom the otherwise frequent attacks ceased entirely during a fairly fatiguing walking tour in the mountains. Possibly the increased consumption of muscular energy retards the accumulation of the "tension," which eventually leads to the "epileptic discharge."

In regard to the treatment of the disease itself, we shall only rarely, when dealing with "reflex" epilepsy (*vide supra*), or where the attacks are caused by other diseases, have to consider ætiological conditions. The excision of old scars, the removal of foreign bodies in cases of traumatic epilepsy with any permanent changes in the skull or the brain-cortex, and trephining, are said to have brought about permanent recovery in a few cases. We should also deal with any nasal or aural disease in order, by rational treatment of any such trouble, to cause a diminution in the frequency of the attacks. If we discover any intestinal worms, they should be expelled. In genuine typical epilepsy we have no such indication to fulfill, and we must have recourse to such treatment as experience shows can improve symptomatically the expression of the disease.

Among these remedies the bromin preparations, particularly potassium bromid, have an undisputed right to the first place. They should be the first remedy tried in any severe case. Apparently they act by directly lowering the irritability of the cortical motor centers. Rather large doses are requisite. Beginning with about ʒss. (gm. 2 to 3) a day, we may find it advisable to increase up to ʒj or jss. (gm. 5 to 6). The bromid may be prescribed in water (1 to 10 or 15), or in powders, which the patient himself is to dissolve in a glass of water, seltzer, or sweetened water, if desired. It should always be taken in a good deal of water to avoid irritating the stomach. The total amount for the day is usually divided into two or three portions, but the whole may be dissolved in a large amount of water (seltzer water) and drunk gradually through the day. The other bromid salts, the bromids of sodium and ammonium are also frequently employed. Strontium bromid has also been recommended, but it appears that it can be dispensed with entirely. Sodium bromid has the advantage of disturbing the stomach less than does the potassium salt. It is well to combine the various bromids. Erlenmeyer recommends a mixture of 2 parts each of potassium and sodium bromids with 1 of ammonium bromid in so-called "bromin" water, or put up as effervescent bromids. Bromipin (bromids dissolved in oil of sesame ℥ss. to ℥j [gm. 15 to 30] *pro dosi*) is highly recommended, as it causes fewer unpleasant symptoms than the bromid salts. Bromglidin is a combination of bromids with vegetable albumin.

In using the bromids, persistence is necessary for at least months, and often, with occasional interruptions, for years, if benefit is to be obtained. It is a good plan to use the remedy according to some regular scheme—e. g., commence with gr. xxx (gm. 2) daily, increase every week by gr. xv (gm. 1) up to about ʒj to ʒjss. (gm. 5 to 6) or even more daily, and then again reduce. In case there are unpleasant symptoms due to the remedy, such as excessive acne, a bad breath, muscular lassitude and tremor, cardiac weakness, dyspepsia, impotence, or mental depression and weakness of memory, we must diminish the dose, or even omit the medicine for a time. Many patients are greatly annoyed by pustules due to the bromid; this can sometimes be avoided by giving Fowler's solution at the same time.

Some physicians (Toulouse and others) have recently attached great importance to reducing the consumption of ordinary salt (sodium chlorid) to a minimum during the bromid treatment of epileptics. This is accomplished either by a strict milk diet or by not adding any salt to the food. In cutting out the salt, the bromid to a certain extent must take its place, and therefore the efficacy is said to be much greater when the food is deficient in salt. The necessary therapeutic dose of bromids is consequently said to be very much less (not over gr. xlv [gm. 3] potassium bromid daily) with a salt-free diet than with a regular diet. I have myself treated a series of epileptics according to this method, and have seen apparently good results in some cases, while in other cases particularly favorable therapeutic results were not obtained. I can therefore recommend a trial of the "Toulouse method," although I do not agree with the exaggerated praises of other authors. If the attacks are decidedly abated during the bromid treatment, the dose may be gradually diminished, to be increased again, however, if there be any tendency to a relapse.

There is no doubt that the frequency of the attacks can be lessened by the continued use of bromids. This action is, of course, manifest only in those cases where the individual attacks occur rather frequently (every three or four weeks or oftener). If we are treating epileptics who have intervals of six months to a year or more between the individual attacks, it is hardly possible to decide with certainty whether the bromid treatment has any efficacy, and the long-continued use of bromid which is necessary is often attended by unpleasant accompaniments. We are therefore usually disposed in such cases to abstain wholly from the bromid treatment. Moreover, during the continuous administration of bromids, careful observation of the action of the drug on the general condition of the patient is necessary for each individual case. Flechsig recommends combining the bromid treatment with the use of opium. We should give at first simply the extract of opium, beginning with gr. ijss. (gm. 0.15) a day, and gradually increasing to gr. vj–viij (gm. 0.25 to 0.35) or more, always divided into two or three doses in the day. After using opium for six weeks, we stop it abruptly and begin with a bromid treatment, continuing about two months. The reports of the results obtained with this method differ widely. It is not generally accepted.

Recourse to other remedies is seldom had, unless potassium bromid fails, or for some cause must be discontinued. We may then try valerian in powder, gr. viij to xxx (gm. 0.5 to 2) several times a day, or as an infusion of 15 or 20 parts of valerian in 150 of water. It is a very good plan to give patients who are taking bromid a cup or two of cold valerian tea at bedtime. Belladonna may also be exhibited in pill form, gr. $\frac{1}{6}$ each of the extract and the leaves, giving two to six of such pills daily, gradually increasing the dose; or a pill of atropin, gr. $\frac{1}{120}$ (gm. 0.0005), three to five times a day; or zinc oxid in the dose of gr. j to iij (gm. 0.05 to 0.20), perhaps combined with gr. j (gm. 0.05) of extract of belladonna [U. S. P.], and gr. xv (gm. 1) of valerian, as a powder, three times a day, and finally chloral in small doses (gr. v to viij a day [gm. 0.3 to 0.5], divided into several doses). Combinations of the bromid salts with some one of the remedies mentioned have seemed to us very advantageous in many cases. I have sometimes seen a decidedly favorable action from a mixture of the bromids with small doses of chloral or with atropin, especially where there are frequent rudimentary attacks. In epileptic attacks appearing late in life, chloral also has repeatedly seemed to me to be particularly efficacious. Bechterew recommends the combination of bromid with cardiac tonics, digitalis or adonis vernalis (an infusion of 2 or 3 parts of adonis vernalis in 180 parts of water, to which 8 to 12 parts of bromid are added). Many remedies formerly recommended are not worth mentioning, as they are wholly inefficacious. [It is often of advantage to prescribe thorough catharsis weekly as a routine treatment.—K.]

Electrical treatment (careful galvanization of the head and sympathetic) is on the whole little used in epilepsy. However, a rational general treatment of the patient with baths (salt baths, CO_2 baths), hydrotherapy (friction, half baths), electric-light baths, etc., is to be recommended in addition to the medicinal treatment, and occasionally in place of it. Severe cases are treated preferably in a well-conducted institution.

Finally, we must say a few words on the surgical treatment of epilepsy. It is indicated in the first place in cases of symptomatic epilepsy if the attacks are frequent, and not influenced markedly by internal medication (bromid), provided that the attacks are in all probability referable to a circumscribed affection of the motor cortex. Suitable cases of traumatic epilepsy have the best prospects for recovery; after these, the cases of Jacksonian epilepsy developing after encephalitis, etc. In cases of genuine epilepsy, Kocher, F. Krause, and others have attempted by making a valve in the skull (i. e., the removal of a bone flap, paring it down around its edges and then replacing it), permanently to reduce the pressure on the brain, and consequently to diminish the frequency of the attacks. A few good results have been obtained among many failures, so that we are justified in recommending this method in grave and otherwise hopeless cases.

Treatment during the Paroxysm.—In most cases we can do little during the seizure except to take such precautionary measures as common sense suggests. We possess no means of suppressing an attack when once under way; nor, indeed, is it often dangerous. In individual instances the patient finds out from experience some method to cut short the paroxysm during the aura. For instance, there are cases where tightly bandaging or vigorously rubbing the part in which the aura originates will avert the convulsions. A number of cases have also been known where the ingestion of a generous quantity of common salt during the aura has had the same effect. In these cases the aura usually starts from the epigastrium. One of our patients, whose attacks began with a feeling of rectal tenesmus, maintained that she could almost invariably suppress the convulsions by promptly going to stool, if she had time and opportunity. It was formerly a frequent maneuver to seek to ward off the attack by compressing the carotids, but this usually fails. Berger recommends the inhalation of nitrite of amyl at the commencement of the fit, having repeatedly seen benefit follow its use.

In the " status epilepticus," narcotics are the most deserving of trial, and in particular chloroform or ether given by inhalation, and the internal use of chloral (gr. xlv to lxxv a day [gm. 3 to 5]). Amyl nitrite may also be of service.

APPENDIX

INFANTILE CONVULSIONS (ECLAMPSIA INFANTUM)

CONVULSIONS in childhood are of such frequency and importance as to justify brief special mention here.

Every practitioner learns from daily experience that the young are especially predisposed to convulsions. Probably this is due in part to excessive reflex excitability of the brain in childhood. Thus, children not infrequently undergo convulsions under circumstances in which adults would very rarely have them. They sometimes are seen in children in the beginning of acute febrile diseases, such as pneumonia, scarlet fever, measles, febrile sore throat, etc. They also occur from indigestion, particularly when the stomach has been overloaded; sometimes on account of teething, or because of intestinal worms. In these cases they are usually regarded as of reflex origin, although they may possibly be due to the action of toxins (especially with intestinal worms).

Convulsions may occur in very early life without ascertainable cause. In isolated cases they are really the commencement of epilepsy, as is seen afterwards. Again, they may be due to some actual lesion in the brain. For example, if one recalls the initial stage of the acute poliomyelitis and acute encephalitis of children (*vide* pages 400 and 524), it will not seem improbable that many rapidly fatal cases of " convulsions " are really instances of the diseases mentioned. This point has not yet been at all satisfactorily investigated by pathologists. Often convulsions occur suddenly in children and then cease, never to recur, without our being able to find any explanation of the attack. It is possible that we have frequently to do with auto-intoxications from the gastrointestinal tract. Experience shows that rachitic children are especially liable to suffer from eclampsia—possibly because of cranial rachitic changes (?). Not infrequently the eclamptic convulsions are the sign of an individual morbid increase of the general excitability of the nervous system (so-called spasmophilic tendency). This spasmophilic condition is shown also in the susceptibility to attacks of tetany (*q. v.*), spasm of the glottis, in the increased electrical and mechanical excitability of the nerves (facial phenomenon, Trousseau's sign). At any rate the occurrence of an eclamptic attack in a child is frequently, though not invariably, the sign of an abnormal constitutional nervous predisposition. For this reason, children who have suffered from eclampsia frequently show later on symptoms of neurasthenia, hysteria, mental deficiency, etc.

The symptoms of the eclamptic attacks are on the whole analogous with those of epileptic paroxysms. The child's eyes become staring and fixed; tonic spasms occur in the face, trunk, and extremities, which, after a few moments, are succeeded by general clonic convulsions. The attack, which never lasts longer than a few minutes, is associated with convulsive respiration, cyanosis, and sweating. Then consciousness returns. A postepileptic sleep does not often occur. The frequency of the attacks is subject to the greatest variations. Occasionally only isolated attacks occur; in other instances they may recur for days with but slight remissions. In such cases the prognosis is dubious, particularly if the child be weakly, but it is by no means absolutely bad. The cause and the significance of the convulsions can seldom be determined immediately.

Symptomatic treatment consists of applying cold to the head, wet packs, sinapisms on the chest and the calves of the legs, and perhaps an enema (to which vinegar may be added). These measures generally answer for mild cases. If the fits are very frequent and violent, we may allow even small children to inhale chloroform, often with great advantage. A dessertspoonful is to be poured upon a handkerchief and administered cautiously. In addition, sodium bromid and enemas of chloral hydrate, gr. vijss. to xv (gm. 0.5 to 1) may be tried.

Recurrence of the attacks is best avoided by a proper regulation of the feeding of the child (mother's milk, etc.). If there are indications of rickets, the administration of cod-liver oil with phosphorus should be tried.

[A bath at a temperature of 90° to 95° F., while cool water is applied to the head, seems often to be of service; if the child be exhausted by diarrhea, the cold to the head should be omitted. In a teething child it can do no harm, and it sometimes has a very marked beneficial effect to lance the gums thoroughly. Enemas containing chloral with or without bromid of potassium are more used in this country than is chloroform by inhalation.—S.]

CHAPTER V

CHOREA

(*Chorea Minor. Sydenham's Chorea. St. Vitus's Dance*)

Ætiology.—Centuries ago the name *chorea* (dance) was applied mainly to those strange states of " dancing mania " which were endemic in certain places, being due to excessive mental excitement and to the innate propensity to imitation. The specific for this condition was held to be a pilgrimage to some shrine of St. Vitus. At the present time, however, chorea is used to designate a perfectly definite disease, of which the characteristic symptom is the appearance of certain peculiar motor phenomena due to irritation of the nervous centers. It is sometimes called *chorea minor*, in contradistinction from what was formerly termed *chorea major* or *magna*. This latter is, however, not a genuine, independent disease, but a manifestation of hysteria (*q. v.*), or apparently in many instances of epilepsy.

Chorea minor is mainly a disease of childhood. It occurs most often between the fifth and fifteenth years, although it may be seen both earlier and later. There is a slightly greater liability to it in girls than in boys. Hereditary neuropathic tendencies are also a factor in its ætiology, but not a very important one. (See the next chapter for Chronic Hereditary Chorea.)

As to causation, in many cases nothing definite can be made out. Mental excitement, as from fright, seems in some few instances to favor the onset of the trouble. It is also certain that the imitative impulse will often lead to choreic movements in healthy children who come in contact with choreic patients, but it is doubtful whether this " imitative chorea " can be regarded as true chorea. It seems to us far more correct in such cases, which are very amenable to psychical treatment, to speak of an hysterical chorea. There is a very interesting connection between chorea and acute articular rheumatism. Although the statement of certain authors, that almost every case of acute articular rheumatism in children is followed by chorea, is far too strong, yet this sequence is comparatively frequent. Chorea is sometimes seen also in children who have a mild form of chronic rheumatism, or especially in such as have valvular cardiac disease, whether preceded by articular rheumatism or not. This undoubted and close connection existing between chorea, articular rheumatism, and valvular heart disease, indicates the possibility that in true chorea there is an intoxication arising from some infection. It is to be recommended, therefore, even in apparently idiopathic cases of chorea, to look for some preceding slight infection (sore throat, etc.).

Pregnancy exerts a special influence upon the development of chorea in women. *Chorea gravidarum* is most frequent in comparatively youthful primiparæ. It occurs most frequently in the third to the fifth month of pregnancy. The intimate causal relation between chorea and pregnancy is shown by the fact that, after the spontaneous or artificial termination of the pregnancy, the chorea soon ceases. The nature of the relation between chorea and pregnancy can be made clear only by assuming that there is some toxic factor present, just as in the nephritis of pregnancy and puerperal eclampsia.

Clinical History.—Chorea usually begins gradually and without any special prodromata. Sometimes, however, there are prodromal symptoms, chiefly a certain mental depression and irritability, with indisposition to intellectual effort. There may be slight rheumatic pains or anorexia, and other evidences of constitutional disturbance.

Ordinarily the peculiar motor disturbances are the first thing to attract the attention of the patient or its parents. There are involuntary and irrepressible movements in the most diverse groups of muscles. Both single contractions and also complicated movements occur independently of the will, and in all parts of the body, now in one place, now in another, sometimes in a single member, and sometimes in several at once. The movements may be made in rapid succession, or may be separated by long intervals of quiet. The facial muscles may be involved, causing an occasional wrinkling of the brow or distortion of the mouth. When there is marked chorea of the facial muscles, as usually happens in severe cases, the patients often make the strangest grimaces, sometimes with some particular expression of emotion (laughter, anger, etc.). The eyes also may take part in the involuntary movements, being moved from side to side, or opened and closed. The pupils are frequently dilated. If the patient be asked to protrude his tongue and keep it quiet, it will often be involuntarily withdrawn into the mouth or thrust to one side. With marked chorea of the tongue, speech is noticeably affected. It is explosive and indistinct. Many children consequently refrain almost entirely from talking. The laryngeal muscles have also been observed to make choreic movements. The arms are frequently the most affected of any part; they are twisted, flexed, elevated, put behind the back—in short, moved in every conceivable way. The trunk is generally but little disturbed in the milder cases, but in severe ones the whole body participates. The patient stands up, lies down again, turns upon his side, etc. The legs are seldom as much disturbed as the arms and face, but slight movements of the lower limbs are very frequent; the foot is thrust forward or extended, the knee is flexed, and so on. In general it may be said to be characteristic of chorea that the abnormal motor irritation usually affects a considerable number of muscles simultaneously, thus exciting all sorts of combined movements; and, secondly, that choreic movements, for the most part, are not short twitches, but take place in a manner decidedly similar to that of voluntary movements.

The severity of the movements varies greatly in different cases. At first they may be too slight for the unpracticed eye to catch. Many children in an incipient stage of chorea are unjustly punished at school because they write badly or do not sit quietly. Many cases are mild throughout, never having very severe disturbance. Others, though considerably annoyed, can, nevertheless, walk or stand alone. In the severest cases, however, the whole body is continually in active motion. The patient throws himself about in bed, and all the extremities exhibit constant violent and irregular movements. The ingestion of food is extremely difficult, sleep is disturbed, and the patient's flesh and strength are rapidly and greatly diminished.

Further, each individual case presents variations in severity at different times. If the patient be left quietly to himself, the contractions are comparatively very slight. As soon as he is conscious of being watched, or as soon as anyone speaks to him, his condition usually becomes much worse. On the other hand, of course, the movements may also be somewhat influenced by the will. The patient sometimes becomes quieter if the body is completely supported and gently restrained, etc. During sleep the choreic movements usually cease altogether.

In many cases all the voluntary muscles are involved, but sometimes the disease is limited to certain groups of muscles. Very frequently the disturbance is mainly unilateral (hemichorea); the opposite side of the body then exhibits few involuntary movements, or, it may be, none. As already stated, the face and upper extremities are often more affected than the trunk and lower limbs.

These motor disturbances often constitute the sole or the predominant symptom of chorea. There is hardly ever muscular weakness or paralysis. It is remarkable how little feeling of fatigue there usually is, despite the incessant activity. In a few cases only of genuine chorea have we seen actual paresis, affecting, for instance, one arm, or in hemichorea the choreic half of the body. Sensation is unimpaired. The reflexes do not exhibit striking peculiarities. The tendon reflexes we have usually found normal, more rarely very weak, or even a little increased. Marked hypotonia of the muscles is occasionally associated with the diminution of the tendon reflexes. Through the diminution of the muscular tonus, the limbs become flaccid and apparently paretic (so-called chorea mollis). There may be isolated spots in the spinal column tender on pressure, but this is not at all constant. That chorea may be complicated by arthritis and valvular cardiac disease has already been stated. Some caution should be exercised in making a diagnosis of cardiac lesion here, for experience shows that choreic patients are apt to have functional murmurs and slight arrhythmia of the cardiac action. [Osler has found that the endocarditis of chorea is very apt to lead to organic valvular disease. Of 110 choreic patients that he examined more than two years after the attack, 54 presented signs of organic heart disease.—K.] The temperature is not elevated, in spite of the constant muscular contractions, nor is the amount of urea excreted by the kidneys increased.

Slight mental disturbance is frequently observed. The patient is often rude, peevish, capricious, incapable of mental exertion, irritable, and inclined to tears. More marked mental disturbances (confusion, hallucinations) are sometimes met with in the severer cases, especially in chorea gravidarum. [These mental disturbances sometimes become very severe (*chorea insaniens*). The patient becomes excited and delirious, and is very apt to die in a few weeks of general exhaustion, with considerable fever, 104° to 105° F.—K.]

The entire process generally occupies several months. In mild cases, however, recovery may ensue at the end of a few weeks, while, on the other hand, cases may last a year or even longer. Variations in the intensity of the chorea are often witnessed. These are sometimes spontaneous, and sometimes are due to outward influences. Even when the disease has apparently disappeared, we must be prepared for a possible relapse. The disease may appear repeatedly during several years, in such a way that it is not easy to determine whether the different attacks are relapses or new illnesses. The protracted cases are, as a rule, comparatively mild, and many cases that begin with great violence end comparatively early. In adults, and still more frequently in the aged (chorea senilis), however, the chorea sometimes assumes a chronic form and

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gradually increasing to 8 or 10 drops. [In severe cases larger doses may be given. It should be pushed up to 20 drops or more, stopping for twenty-four hours if symptoms of poisoning appear, and beginning again at the same dose. It should be freely diluted.—K.] In children under six the dose should be made somewhat smaller. If the child be anæmic, iron may be given in addition; or, if there be great restlessness and loss of sleep, narcotics may also be administered. Antipyrin, in doses of gr. vij to xv (gm. 0.5 to 1) several times a day, is sometimes of decided service in severe cases. We give it especially in cases associated with rheumatism. Many observers have seen good results from sodium salicylate, gr. xv to xxx three times a day (gm. 1 to 2), or aspirin, gr. v to viij (gm. 0.3 to 0.5) several times a day. If these remedies have no effect we may also try sodium bromid or bromid of potassium in large doses, ʒss. or more (gr. xxx to xlv [gm. 2 to 3]) a day. In severe chorea we have seen frequently undoubted benefit from the bromids. [Sulphonal, gr. iij to v (gm. 0.2 to 0.4) three times a day, acts well, as an adjuvant to arsenic, in cases where there is much excitement. Exalgin, gr. ij to iij (gm. 0.1 to 0.2) three times a day, has also been recommended.—K.] Narcotics should be employed very cautiously in chorea. Although chloral has been recently recommended for grave cases, there are instances known where this remedy has been followed by unfortunate results; but we may try the continued and cautious use of small doses of chloral, giving to children gr. ij to v (gm. 0.1 to 0.3), and to adults gr. viij to xv (gm. 0.5 to 1) daily, or a few larger doses at night. In grave cases of chorea in adults, careful injections of morphin are occasionally attended by noticeably good results. Injections of hyoscin hydrobromate, gr. $\frac{1}{120}$ to $\frac{1}{60}$ (gm. 0.0005 to 0.001) daily have also occasionally a very quieting effect.

Hydrotherapeutics of a mild kind do good, and can easily be carried out in most instances. Thus, we may use lukewarm baths, a wet pack, or gentle sponging with water at 72° to 82° F. (18° to 22° R.) to great advantage. In some obstinate cases sweating seems of service. We have also repeatedly seen decided benefit from the use of methodical gymnastic exercises, consisting of regular flexion and extension of the arms, marching in time, etc. Electrical treatment (weak galvanization of the back and head) is of no great importance.

In the chorea of pregnancy, which sometimes is a very violent disease, the remedies previously mentioned may be employed. If they do no good, artificial delivery may be required; after which, as we can attest, there is generally a rapid abatement of all symptoms.

CHAPTER VI

CHRONIC HEREDITARY CHOREA. ELECTRICAL CHOREA. PARAMYOCLONUS (MYOCLONIA)

1. **Chronic Hereditary Chorea (Huntington's Chorea).**—Chronic chorea also manifests itself by severe choreic movements of the body, but in its nature it is a wholly different disease from ordinary chorea. It is notably of family (generic) or hereditary occurrence. The symptoms of the disease begin, as a rule, between thirty and forty. The choreic movements gradually become more

severe and increase to the most curious and strange movements of the face and the whole body. The ability to walk and to use the arms in eating, dressing, etc., is retained for a long time, but every action is accompanied by extremely conspicuous associated movements and twitchings of the whole body. The choreic restlessness is increased by mental excitement and by prescribed voluntary movements. If the muscles of the face and tongue be involved, speech is disturbed. All movements are performed hastily and in a jerky manner, because the patients must time the execution of their movements to the brief intervals of rest. The sensibility and the other nervous functions are unaffected. The tendon reflexes are generally fairly lively. Mentally, however, we notice a decided but gradual failure as time goes on, which may increase to complete dementia. In other cases, periods of marked mental excitement or melancholic states occur. The latter may lead to suicidal attempts. The anatomical basis of this remarkable disease is still unknown. In the cases which have come to autopsy we, however, find, as a rule, thickening of the meninges, atrophic conditions of the cerebral cortex, proliferation of the neuroglia, miliary encephalitic foci, etc. The significance of all these changes is, however, still extremely uncertain. As has already been stated, the prognosis is hopeless, for the disease is incurable. The remedies used in ordinary chorea never mitigate the symptoms.

2. **Electrical Chorea.**—Henoch gave this name to a form of chorea in children which differed from the ordinary chorea by the much quicker, lightning-like contractions coming on in single muscles (especially of the neck and shoulders). The contractions give the impression that the muscles have been put in contraction by single galvanic shocks. They come on at rather long intervals of three to five minutes. This variety of electrical chorea is evidently closely related or identical with myoclonia, which will be subsequently described. The same name, "electrical chorea," has also been given to a peculiar disease, said to be endemic in Lombardy, first described by Dubini, and which is therefore known by the name of "Dubini's disease." In this affection also, after preceding pains in the head and back, rapid, jerky, muscular twitchings occur, usually beginning in one arm and gradually involving all the extremities. After several weeks or months paralysis and muscular atrophy set in, and not infrequently epileptiform convulsions and fever. The skin is hyperæsthetic, so that at every contact of the skin lively twitchings result. The sensorium remains clear. The disease frequently ends fatally after a few weeks or a longer interval of time, with the symptoms of cardiac paralysis, or with the onset of a comatose condition. Recovery ensues only in a small percentage of the cases. Nothing definite is known as to the anatomical changes.

3. **Paramyoclonus Multiplex (Myoclonia).**—This name was first used by Friedreich for a case observed by him, in which there were clonic spasms in symmetrical groups of muscles of the arms and legs, which came on in paroxysms without any disturbance of consciousness. There was also a considerable increase of the tendon reflexes. Such cases have since been described in considerable numbers, but undoubtedly very different morbid conditions have been confounded with one another. We can regard as genuine paramyoclonus—that is, as a specific disease *sui generis*—only those cases where lightning-like contractions come on in single muscles, often without any visible effect of

movement. The muscles of the trunk and extremities are most frequently involved. This peculiar morbid condition is on the one hand most closely allied to electrical chorea mentioned above, while, on the other hand, it has a decidedly close relation to the different forms of *tic convulsif* (clonic facial spasm, clonic accessory spasm, *q. v.*). A large number of the cases described as myoclonia or paramyoclonus, however, are undoubtedly to be classed as hysteria (*vide infra*). By considering carefully the ætiology and the course of the disease, we can generally readily distinguish the curable hysterical forms of myoclonia from true chronic myoclonia, which is evidently a grave nervous malady of endogenous origin, and closely related to chronic chorea and the chronic tics.

At times these suddenly appearing convulsive twitchings are of a much more complicated form—the muscular twitchings are associated with peculiar sounds uttered in a spasmodic manner, or even with the forced interjection of words. If these words are of an objectionable character, we speak of so-called coprolalia. If there is a forced repetition of words which are heard, the condition is called echolalia. All these peculiar symptoms are combined in different ways in the peculiar clinical picture called by the French authors "maladie des tics." The disease is intimately associated with certain forms of hysteria, which is particularly apparent from the good results obtained by properly conducted psychical treatment.

Finally, we have still to mention a special variety of myoclonia described by Unverricht, Lundborg, and others. It is characterized by its occurrence in various members of one family, by the extension of the twitchings to the lingual and pharyngeal muscles, and by its complication with epileptic attacks, which at times are grave, and may occur frequently. The condition not infrequently terminates in marked dementia.

CHAPTER VII

PARALYSIS AGITANS

(Shaking Palsy. Parkinson's Disease)

Ætiology.—In 1817 Parkinson described for the first time a disease which he named "shaking palsy." It is not of very frequent occurrence, and as yet little has been ascertained with regard to its ætiology. In most cases it develops very gradually, without any demonstrable cause. It almost always attacks elderly persons, being very rare before the thirty-fifth year. Sex does not seem to exert any great predisposing influence. Hereditary neuropathic tendencies can indeed be traced in some instances, but they are certainly of less potency in paralysis agitans than, for instance, in epilepsy. Special exciting causes have sometimes been observed, such as catching cold, violent emotional excitement, and traumatic influences (blows, falls, injuries, etc.). Berger reports two cases where the first symptoms appeared after an acute febrile disease (typhoid fever).

Clinical History.—Paralysis agitans has two characteristic symptoms, viz.: (1) Peculiar evidences of motor irritation, evinced by tremor, and (2) a condition of stiffness and persistent shortening of certain muscles, consequent upon which is a series of peculiar motor phenomena.

The trembling is generally the earliest symptom to attract the patient's attention. It usually begins in the hands, especially in the right hand, and then gradually involves the arm and leg on the same side, next the other arm and leg, and finally, in well-marked cases, the entire body. In some cases the symptoms may be for some time limited almost entirely to one half of the body.

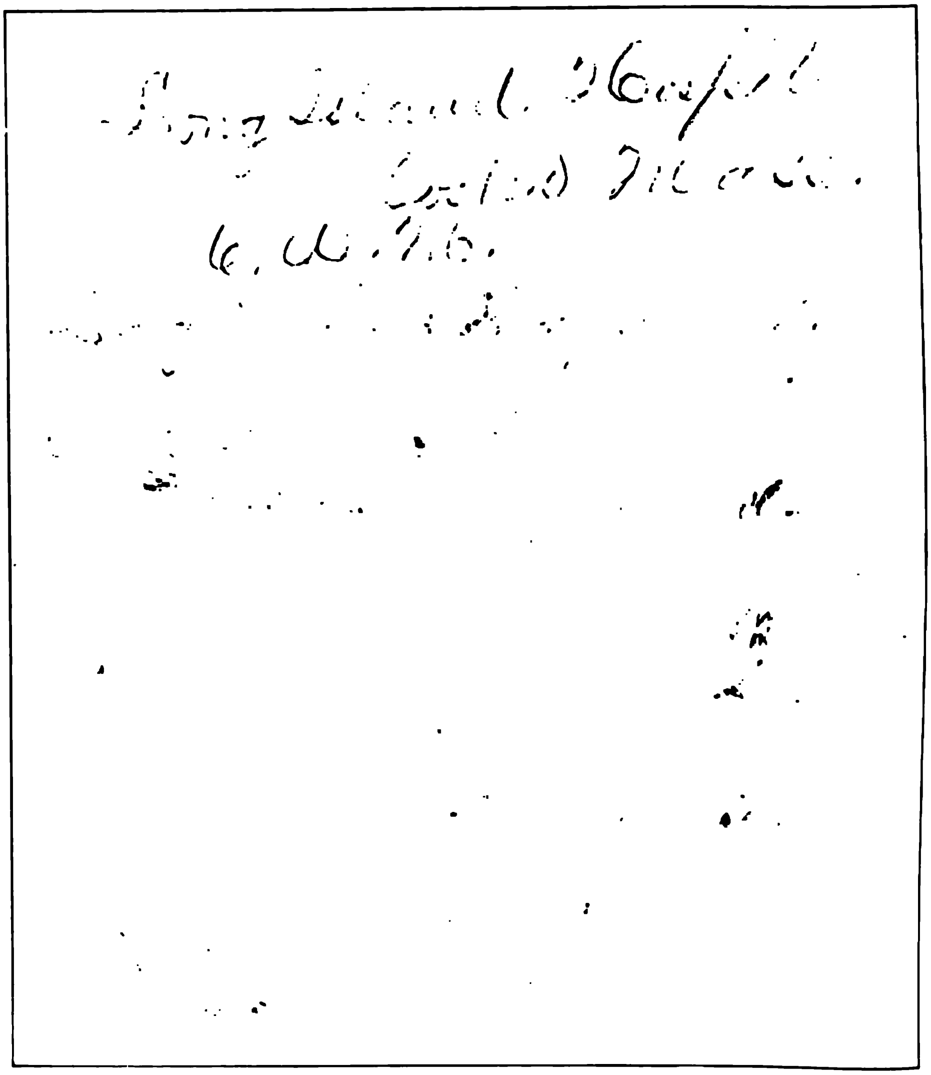

FIG. 215. Handwriting of a patient with paralysis agitans at the Long Island Hospital, Boston, showing increase of the tremor from hour to hour.

The form of tremor is very characteristic. There are rapid, uniform, oscillatory movements of varying extent. The tremor is usually greatest in the hands and arms. At the same time the thumbs and half-flexed fingers exhibit a movement which suggests spinning or pill rolling. The forearm is generally flexed and extended in rapid alternation, but it is always very difficult to determine just what muscles contract. [The tremor often gives the handwriting a very characteristic appearance (see Fig. 215).—K.] With regard to the trunk, it is often a question whether its tremor is of independent origin, or due merely to the motion of the extremities. Charcot states that the head and the facial muscles are never implicated, but there is doubt about this point. We ourselves, as well as other observers, have repeatedly seen independent tremor of the head. As to the face, we have observed that the muscles about the chin seem to suffer chiefly.

The tremor of paralysis agitans is almost continuous. It may, indeed, cease for a moment in a limb, but only to recur immediately. The quieter the patient is in mind and body, the less violent are the movements. If he is excited, or begins to speak, or is watched, the tremor at once becomes exaggerated, and may be violent enough to jar the whole body vehemently. Active motion does not intensify the tremor. On the contrary, it may often be observed that the trembling abates when the muscles undergo vigorous voluntary contraction, as when a weight is lifted, or the hand of another is firmly grasped.

The second symptom is almost more characteristic than the first. It consists of a peculiar rigidity of the muscles. We generally notice, even in the face, a peculiar tension of the muscles. This often produces a stolidity of expression, so that the emotions are less clearly depicted than upon the countenance of a healthy person. We have been especially impressed in several cases by the infrequency of winking. The head gradually becomes more and more flexed. When the disease has lasted some years, the chin may even rest upon the sternum. The muscles of the trunk and extremities also stiffen gradually, and lead to peculiar and almost pathognomonic appearances. The body is bent over forward; the arms cling to the trunk, and are flexed at the elbow joint; the fingers are flexed, especially at the metacarpo-phalangeal joint; the thumbs rest against the fingers, as if holding a pen, or else are flexed inward upon the palm, and the legs are somewhat bent at the knee. The accompanying picture (Fig. 216) is from the photograph of a patient who was for a long time under observation at the clinic in Leipsic, and gives a good representation of the characteristic posture.

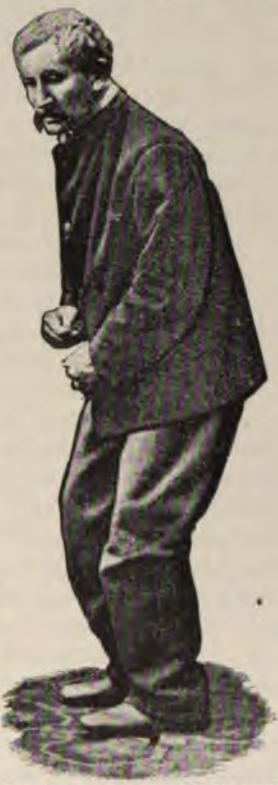
FIG. 216.—Characteristic position of the body in paralysis agitans.

The stiffness of the muscles also operates to impair motion in various ways. In particular, all movements of the trunk are greatly impeded. In advanced cases the patient cannot get upon his feet, if he is lying in bed, without help. Inasmuch, however, as the muscular strength usually remains good (*vide infra*), he requires to exert but a slight traction upon some helping hand in order to attain an erect posture. On the other hand, the patient is often utterly unable to turn in bed from one side to the other. In severe cases, therefore, it is often necessary to alter the patient's position several times in a night, especially as lying long in one attitude makes him feel very uneasy. If the patient is in a chair, he cannot get up of himself, because it is impossible for him to bend his body forward in the necessary manner; but with just a little help he can stand up, and he is then able to walk alone or even to run. Then, since the flexion of the trunk forward brings the center of gravity forward also, and the trunk cannot be sufficiently bent backward, he is apt to "get going," so that he cannot stop until he brings up against a post or a wall. If such a patient, with a considerable degree of anteflexion and rigidity of the trunk, is slightly pushed from behind, he will have to start into a run to avoid falling. This phenomenon is termed "propulsion." A push backward, which brings the center of gravity behind the point of support, is very likely to make such a patient fall, as he will seldom succeed in moving backward fast enough to recover his balance (retropulsion). Both propulsion and retropulsion are conceived by Charcot to be "forced movements" (see page 203) in the strict sense of the term. We are, however, convinced, as the result of numerous observations, that these phenomena can in every case be explained simply by the mechanical conditions arising from displacement of the center of gravity and the muscular rigidity. Again, the reason why many patients are prone to keep their arms behind them as they walk is that such a position contributes slightly toward bringing the center of gravity farther backward.

The movements of the extremities are less impaired than those of the trunk, but they often betray a certain slowness and stiffness. The strength of the muscles may be preserved for a long while, but often there is at last evident paresis. Even in the early stages of the disease the muscles may become easily fatigued. The impairment of facial expression has already been referred to. In many cases the muscles of the eye also seem to participate in the rigidity, making it difficult for the eye in reading to follow rapidly along each line, or to pass from the end of one line to the beginning of the next. [The voice is often affected; the patient talks in a high-pitched monotone.—K.]

Muscular rigidity, as has been said, is almost more characteristic of paralysis agitans than the tremor. Indeed, there would seem to be cases, as we have ourselves seen, where, at least for a time, the posture of the patient is typical, and yet there is no trembling. Such cases might be called *paralysis agitans sine agitatione*. In uncomplicated cases all other nervous functions remain perfectly normal. Sensation is never impaired. Sometimes some pain is felt at the commencement of the disease, particularly in the shoulders. There is no striking disturbance of reflex action or of the bladder. In a few cases of paralysis agitans, cerebral and mental symptoms have been observed; but they are so rare that it is impossible to say whether they belong to the disease or are merely accidental complications. It is also noteworthy that many patients complain of a subjective feeling of excessive warmth. The internal temperature is normal, but it is said that the temperature of the surface of the body is frequently somewhat elevated. Sometimes there is a tendency to excessive perspiration. [Mosse and Banal have found that the excretion of urea and the total excretion of phosphoric acid is increased in paralysis agitans, but that the excretion of imperfectly oxidized phosphoric acid is diminished, thus showing that there is increased combustion.—K.]

The disease runs a very chronic course, perhaps for twenty years or more. From the first, it keeps on slowly but gradually developing. The symptoms rarely exhibit marked alterations of mildness and severity, but for long periods the progress of the disease may be apparently arrested. Recovery has never yet been observed. The final and fatal termination is not brought about by the disease itself, but is due to some intercurrent affection or to general marasmus. The original of the picture on page 605 came to a pitiable end at his own home by tumbling face downward into a puddle of water. He could not get up, and was drowned.

Nature of the Disease.—The true nature of the disease is unknown. Inasmuch as the disorder is purely a motor one, the corresponding lesions must be sought somewhere in the motor system. As yet, however, post-mortem examinations of the nervous system, even with the microscope, have revealed no characteristic changes; at least, we can ascribe no significance to the statements in this respect made by some observers. We must, therefore, confess that we have even had a doubt whether it is justifiable to claim that paralysis agitans is an affection of the nervous system at all. Possibly the disease is purely a muscular affection. Certainly it would not be impossible for abnormal processes in the muscles to excite the tremor and tonic contraction. It is not at all impossible that certain anomalies of the internal secretion (thyroid or parathyroid gland?) produce this morbid condition in the muscles by toxic action, but nothing, as yet, is definitely known on this subject. As has been said, there is at present no ground for deciding this question; we would merely suggest it. [Camp has found the muscular fibers swollen and rounded on cross-section, with an increase of nuclei and atrophic changes in the fibers. The parathyroid glands were also diseased. He concludes that the anatomical basis of the disease lies in the affection of the muscles, and that it is probably a toxæmia, due perhaps to alteration in the secretion of the parathyroid glands. The fact that the disease usually begins in one arm and often affects only one half of the body, however, suggests a cerebral origin.—K.]

Diagnosis.—Any typical case of paralysis agitans can be easily and certainly recognized, often at the first glance. The important factors are the peculiar tremor, the characteristic carriage, and the rigidity of the muscles of the trunk and extremities. It was formerly a difficult matter to distinguish between paralysis agitans and multiple sclerosis; but to-day the peculiarities of the two diseases are better known, and confusion is seldom possible. The character of the tremor varies in the two. In paralysis agitans it persists even when the patient is quiet, and it is decidedly oscillatory. The motion in multiple sclerosis is almost always an intention tremor only; and, what is of still greater importance in distinguishing between them, the general appearances of the two diseases are essentially unlike.

Treatment.—As has already been implied above, we possess no means of controlling the disease. Good may be done by lukewarm baths of considerable duration, friction with cool water, gentle massage of the muscles, and careful galvanization in certain cases. Electric baths may also be tried.

Among internal remedies hyoscin, first recommended by Erb, has the most palliative and soothing influence. It is best given subcutaneously, and we must always begin with very small doses, as otherwise unpleasant symptoms, such as vertigo, headache, etc., may arise. We inject $\frac{1}{200}$ to $\frac{1}{100}$ of a grain (mgm. $\frac{1}{3}$ to $\frac{1}{2}$) in aqueous solution (solution of hyoscin muriate, 1 to 1,000, 7 to 15 minims). If well borne we can cautiously increase the dose. [Atropin and hyoscyamin have also shown good results, especially when given subcutaneously.—K.] Arsenic, ergotin, potassium bromid, and physostigmin may also be tried. Francotte praises the action of sulphate of duboisin (subcutaneously or in pills of $\frac{1}{100}$ gr. [gm. 0.0005] three to six times a day); this also must be used with care. [In a few instances the parathyroid gland has been given without much benefit.—K.]

In most cases the treatment is finally limited to careful nursing.

CHAPTER VIII

ATHETOSIS

In 1871 the American neurologist Hammond described under the name of athetosis (ἄθετος, without fixed position) a peculiar symptom of irritation of the motor centers, differing in a characteristic manner from all other forms of involuntary movements, including the epileptiform and choreic. The movements of athetosis (see page 203) are often very complicated and peculiar. The part affected by them is in continuous unrest. If the facial muscles (usually those of the lower division of the facial nerve) and the muscles of mastication be attacked, the face and mouth are constantly being twisted and distorted. If the tongue suffer, as in one case which we saw, speech is difficult and indistinct. If the muscles of the back of the neck be implicated, the head is usually drawn backward or to one side, and is turned and twisted in all sorts of ways. Most characteristic of all, however, are the movements exhibited by the hand and fingers when affected. The fingers are incessantly being separated, extended, flexed, and intertwined, assuming the strangest positions. The accompanying pictures may serve to illustrate this (Fig. 217). The character of the movements reveals that the interossei must be chiefly involved. It is a very frequent result of the unceasing stretching of the articular ligaments of the fingers that at last the articulations become relaxed to such a degree as to permit of such a hyperextension of the fingers as it is impossible for a healthy person to imitate. The arms are generally less severely affected than the hands; and in the lower limbs the trouble is not often so severe as in the upper. The toes may, however, exhibit motions analogous to those of the fingers.

Although in general the movements are continuous, their vigor frequently varies. Thus, they almost always are aggravated if the patient becomes excited. During sleep they generally cease, although in certain instances they have persisted even then, only being diminished. When voluntary motions are made they ordinarily grow feebler; but they may, on the contrary, become exaggerated, taking the form of sympathetic movements.

We must distinguish between genuine idiopathic athetosis and a symptomatic form, which also occurs.

Symptomatic athetosis is seen in various nervous diseases. The first observations reported by Hammond were, most of them, made in cases of epilepsy, or in severe psychoses, and the like. [In Hammond's original case the athetosis was symptomatic and due to a lesion involving the optic thalamus, the lenticular nucleus, and the most posterior portion of the internal capsule.—K.] By far the most frequent source of the phenomenon, however, is hemiplegia, producing what is known as posthemiplegic chorea, or, rather, posthemiplegic hemiathetosis. This is, to be sure, a very rare sequel to the ordinary hemiplegia of elderly persons, but follows quite frequently cerebral infantile paralysis (*vide* page 524). Some traces of athetosis are seen in a majority of the cases of infantile hemiplegia.

Idiopathic athetosis is rare. Here the peculiar movements are the chief, if not the sole, symptom of disease. A few cases of this sort have been reported where the athetosis began without known cause, and usually was limited to some one region. It attacked elderly individuals who were previously

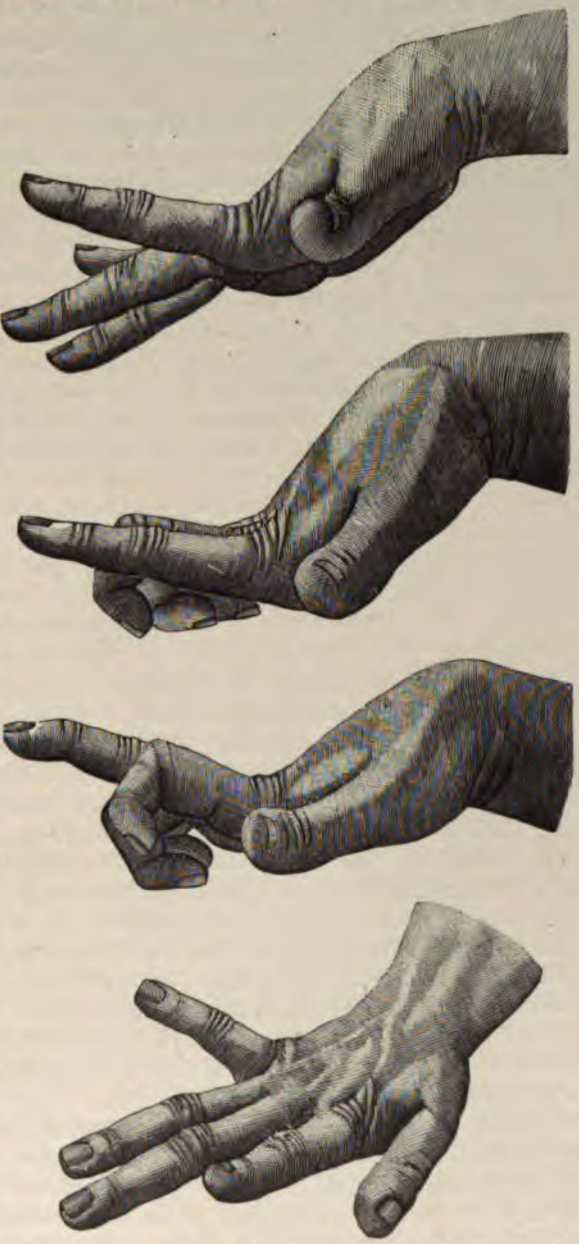

FIG. 217.—Example of the position of the fingers in the movement of athetosis (personal observation).

healthy. Of especial importance is an apparently congenital form of athetosis, dating from the earliest infancy. Of this we have ourselves seen several instances, which closely resembled one another. The condition is a permanent one, not progressive, nor, on the other hand, capable of any great amelioration. The movements are almost always most pronounced in the face, neck, and fingers. Other nervous disturbances, paralysis or impairment of sensation, are entirely absent or are associated with the athetosis in various ways. The intelligence of the patient is sometimes, but not always, diminished.

Of the nature of athetosis, or the locality or nature of the irritation, we possess no information as yet. It is extremely probable that the lesion is in every case a cerebral one. Perhaps it is in the cortex. In symptomatic athetosis we find postmortem the changes caused by the original trouble. In idiopathic athetosis no changes have thus far been reported. In a case of our own which came to autopsy, absolutely nothing abnormal was found in the brain macroscopically. The patient was an elderly female, who displayed typical movements of the arm and hand.

It is not yet known whether recovery is possible. A certain amount of improvement sometimes follows the administration of the bromids or galvanism.

CHAPTER IX

TETANY

Ætiology.—By tetany (the name was suggested by Corvisart) we designate a disease first described in 1830 by Steinheim of Altona and by Dance in France, the chief symptom of which consists of peculiar, intermittent tonic spasms, generally affecting the upper extremities symmetrically. Tetany may appear apparently as an independent disease. Very frequently, however, it is only a concomitant symptom or a sequel of very diverse conditions. The connecting link for all the apparently widely differing conditions under which tetany is observed appears to have been found in certain recent interesting pathological and experimental work. They appear to indicate more and more positively that the real cause for tetany must be looked for in a lesion of the parathyroid glands, or epithelial bodies, as they are now generally called since the publications of A. Kahns. The first hint of this unexpected relation, to which now, as a matter of fact, an entire series of significant observations on disturbances of the internal secretion can be added, was obtained from the observation of N. Weiss, in 1880, that typical cases of severe tetany were not infrequent after removal of goiter. Two Italian investigators, Vassale and Generali, were the first to find that the extirpation of the epithelial bodies in dogs and cats produced distinct tetany, whereas the extirpation of the thyroid gland alone was followed merely by the phenomena of myxœdema (cachexia, etc.). These statements were confirmed by all later investigators (Biedl, Erdheim, Pineles, etc.), and Pineles was also able to demonstrate with comparative certainty that in man, as well, only those cases of removal of a goiter were followed by tetany, in which the epithelial bodies were entirely, or at least in greater part, removed. Finally, fatal cases of tetany in children have been recently repeatedly observed in which, at autopsy, there was indubitable evidence of disease (generally hemorrhage) of the epithelial bodies (Erdheim and others).

Of course, in spite of these notable discoveries, we are still at the beginning of our knowledge concerning the nature of tetany; for, even if we could imagine that a lesion of the epithelial bodies can in some way cause an accumulation of toxins in the body, and in this way produce tetany, we are by no means justified in assuming now that all of our clinical observations on the occurrence of tetany under the most diverse conditions (to which we will refer subsequently) can be positively brought into accord with this one point of view. Nevertheless, we can predict at this time the probable way by which we may finally clearly explain the problem.

Idiopathic tetany is observed in children and young persons between fifteen to thirty years of age. It is a very noticeable fact that it does not occur with equal frequency in all localities. In Heidelberg (Erb, F. Schultze) and Vienna (N. Weiss, Frankl-Hochwart) it is decidedly a common disease, at least at certain times (*vide infra*). In Leipzig, on the other hand, it is one of the rarer diseases of the nervous system, and, aside from the not uncommon cases of tetany in cases of pyloric stenosis, I observed only isolated cases in Erlangen and Breslau also. [It is also very rare in America. Crozer Griffith a few years ago was able to collect only 73 reported cases in the United States, while 40 or 50 cases have been treated at the hospital in Vienna in a single year.—K.] Moreover, in tetany localities, very remarkable differences are observed as regards the season during which cases occur. By far the greatest number of cases occur during the winter and spring months, and principally from January to April. Aside from cases of tetany dependent on the processes of female sexual life, men are affected with considerably greater frequency than women. Although inexplicable, it is preferably among certain callings, above all shoemakers, then tailors, carpenters, and turners that the disease occurs. Isolated cases occur in every calling. Occasionally, also, several cases of tetany occur in the same family or in the same house. All these experiences appear to point to certain, still entirely unknown, infectious causes. Catching cold, occasionally also mental excitement, physical overwork, etc., are assigned as the cause of the first attacks.

Secondary symptomatic tetany is observed most frequently in grave gastric and intestinal diseases, more especially, according to our experience, in benign pyloric stenosis with secondary dilatation of the stomach (Vol. I, page 537). As the attacks are not particularly severe and frequent in these cases, we learn of them only on special inquiry and investigation. In other dyspeptic conditions—viz., cancer of the stomach, appendicitis, cholelithiasis, etc., tetany also occurs. Furthermore, it has been observed in isolated cases in conjunction with acute infectious diseases (typhoid, polyarticular rheumatism, influenza, measles, etc.), and as the result of certain poisons (ergotin, chloroform, alcohol, phosphorus, etc.). The relation of tetany to pregnancy and the puerperal state ("maternity tetany") is of especial interest. Trousseau had already described this variety as "*contracture des nourrices.*" But here, also, the above-mentioned local and seasonal conditions play a part. Symptomatic tetany has been observed in exophthalmic goiter. I myself have seen a very marked case of it. The occurrence of tetany in osteomalacia is interesting, although up to the present time only isolated observations have been reported. Whether the tonic muscular spasms, which occasionally occur in chronic nephritis, are in any way connected with tetany, we will not attempt to decide.

The occurrence of tetany in infancy has still to be briefly considered. It may occur during the second or third month, but is most frequent during the latter half of the first year (Escherich). Poor general hygienic conditions favor its occurrence. Children who are artificially and improperly fed (excessive carbohydrates) are more frequently affected than those who are breast-fed. The intimate relations of tetany to rickets as well as to laryngospasm and to certain forms of eclampsia (*q. v.*) are very marked.

Clinical History.—The paroxysm of tetany usually has certain prodromata, consisting of slight general discomfort and pain, and of a feeling of weakness and stiffness, most marked in the arms. These symptoms last some hours (at least) before the true convulsive stage begins. The extremities, and more particularly the fingers, almost always suffer first; and then, after the arms, the lower extremities become involved. The spasm usually affects the toes before it seizes upon the other parts of the leg. The symptoms are almost invariably bilateral and symmetrical. Exceptionally, the disturbance commences in the leg, or is confined to one side of the body. In most cases the flexor muscles are predominantly affected, giving rise to very characteristic postures (*vide* Fig. 218). The fingers are in apposition with one another, and placed as if holding a pen, or, as Trousseau says, as if the hand were about to be thrust into the vagina during labor, but in the attack the fingers may also be closed in a fist. The hands are flexed, the elbows are also slightly flexed, and the upper arm in severe cases is pressed against the chest. In the lower extremities the toes are flexed, and the feet are in the posture of talipes equinus. The muscles of the thigh are less frequently affected, as are also the muscles of the trunk, the muscles of mastication, the facial and ocular muscles, and the diaphragm. In children, tetany occurs frequently in the form of laryngospasm; in adults, only rarely.

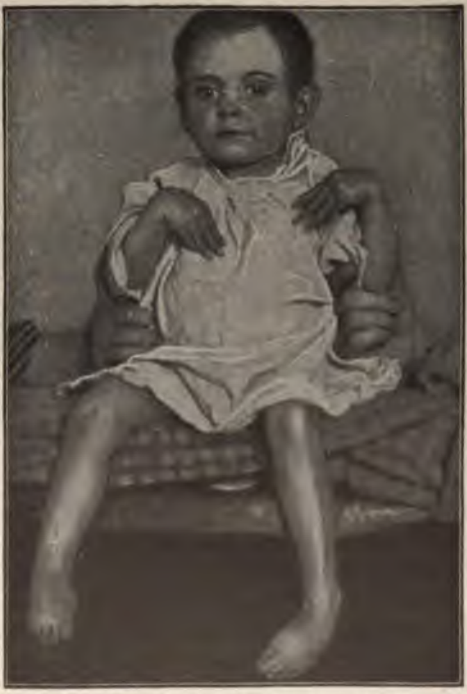

FIG. 218.—Case of tetany in a child (after M. THIEMICH).

The intensity of the tonic spasm is very great. The affected muscles feel as hard as a board, and are usually rather sensitive to pressure. The attack sometimes continues only a few minutes, but not infrequently it may occupy several hours or days.

In children conditions of this kind of continuous tonic rigidity, due to tetany, have been repeatedly observed. Formerly they were occasionally designated as "pseudo-tetanus" or "arthrogryposis." The disease generally develops in a fairly acute manner, and may be associated with fever and severe general constitutional symptoms. The legs are either held rigidly extended or flexed spasmodically on the abdomen; arms, hands, and fingers are generally fixed in a flexed position. In milder cases recovery ensues after several weeks. I have, however, seen two cases with fatal termination.

Other nervous symptoms, in addition to the cramps, are but little pronounced in tetany. The most noticeable are paræsthesiæ, tearing pains, etc. Anæsthesia occurs only rarely. The reflexes are generally normal. An increase or diminution of the reflexes has been found only in isolated cases. If the disease lasts for some time, a certain paretic weakness of the legs may occasionally develop. Myotonic symptoms (*vide* following chapter) have been observed in the muscles of the arm in isolated cases. There is no impairment of consciousness. In only a few cases has a combination of tetany and hallucinatory confusion been seen. In a few instances slight œdematous swelling and other trophic disturbances have been observed, and sometimes profuse perspiration. Optic neuritis has been occasionally observed. The comparatively frequent formation of cataract in tetany is very remarkable. The temperature is normal, or sometimes subnormal or slightly elevated (100.5° to 101° F. [38° to 38.4° C.]), and the pulse is often quite rapid. Polyuria has sometimes been observed.

When the attack ceases, which it always does gradually, and never suddenly, the patient feels perfectly well, save for a slight pain and stiffness in the muscles, but all the muscles often feel weak and unsteady during the whole period of the disease. Even in the interval between the paroxysms there are also some few objective symptoms, which have a most important bearing on the pathology of tetany, and are of the greatest importance for the diagnosis. These symptoms, which, as a matter of fact, must be particularly looked for, are generally designated after their discoverers, as Trousseau's, Erb's, and Chvostek's signs. Trousseau's sign, which is found in most, though not in all, cases is this—a fresh paroxysm can at any time be artificially excited by pressure upon the larger arteries and nerves of the arm, particularly the median nerve and the brachial artery in the bicipital groove. It is not definitely known how compression accomplishes this; but compression of a nerve seems to be the main point, as the corresponding experiments on animals show, in which an artificial tetany is produced by extirpation of the thyroid. We are probably, however, not dealing with a direct, but with a reflex, stimulation. Trousseau's sign is best obtained by tying an elastic rubber bandage around the upper arm, so that a distinct cyanosis is obtained in the peripheral segment. If the test is a successful one, the attack of tetany ensues in a few minutes. Erb's sign is the increased galvanic (much more rarely, also faradic) excitability of the nerves and muscles discovered by this author to be almost always present. The ulnar nerve is preferably tested, and one finds here that the weakest currents are sufficient to produce very lively contractions. The ready occurrence of anodic opening and anodic closure tetanus is also characteristic. Mann and Thiemich attach special importance in children to the occurrence of cathodic opening contractions even with weak currents. Hoffmann and others have demonstrated a similar increased excitability for the sensory nerves and the nerves of special sense (auditory). The third symptom is Chvostek's, which is practically perhaps the most important, because it is the most readily elicited. It consists of an extremely marked increased mechanical excitability of the nerves. The facial nerve is commonly tested. In many cases stroking of the cheek with the handle of a percussion hammer is sufficient to produce twitching of the facial muscles (F. Schultze); in other cases a light tap on the point of exit of the nerves or on their branches is all that is required. Marked excitability of the facial nerve of this sort may, of course, also be found in isolated cases in healthy persons, and in other diseases of the nervous system. Nevertheless, Chvostek's symptom is an extremely characteristic phenomenon of tetany.

The frequency of the attacks varies greatly in individual cases. As a rule, there are several paroxysms daily, but the intervals may last for days, or again may be almost inappreciable. The entire duration of the disease is generally several weeks, but many cases last for months or even years. It is noteworthy that when the paroxysms grow less frequent and violent there is also a gradual diminution in the hypersensitiveness of the nerves and in the reaction to Trousseau's test. As long as these symptoms persist, spontaneous attacks are also possible. Many persons seem especially disposed to tetany, so that the affection repeatedly recurs in them.

The termination of tetany is, as a rule, favorable, but in some cases death results, which is due partly to complications and partly to the severity of the case itself. In children the disease may have an unfavorable termination if the spasms involve the diaphragm or the laryngeal muscles. The danger of "arthrogryposis" has already been alluded to above.

Diagnosis.—The diagnosis is not difficult if we only consider carefully the symptoms presented, the nature of the paroxysms, and the other phenomena above enumerated. It is particularly important in the interval between the attacks to determine the existence of the so-called spasmophilic condition (*vide supra*) by the demonstration of the increased mechanical and electrical excitability of the nerves as well as of Trousseau's sign. It is of fundamental importance, after determining that the attacks are tetany, to discover, if possible, their special cause, and especially to determine whether we have to do with "idiopathic" or symptomatic tetany. It should also be said that tonic spasms, resembling tetany, may also occur in the hands in hysteria.

Treatment.—The main treatment, besides general hygienic measures (especially rest and nursing) and the consideration of all discoverable causes (gastric and intestinal diseases, chronic intoxications, excessive work with the hands, etc.), is electricity. The stabile current is passed upward through the nerves affected; the galvanic current is also applied to the spinal cord, and the anode is applied to the various nerve trunks, with the cathode on the sternum. Dietetic treatment (mother's milk) is of the greatest importance in the treatment of tetany in infants. Internal remedies, such as bromid of potassium, arsenic, and belladonna, rarely produce brilliant results. Morphin and chloral cannot be dispensed with entirely in severe cases. Tepid baths and the cautious use of cold sponging, with friction and massage, may be tried.

The repeated attempts which have been made to cause an improvement of tetany by the administration of parathyroidin are very interesting. Some good results have been already reported, but, on the whole, they are not as yet very remarkable.

CHAPTER X

CONGENITAL MYOTONIA

(Thomsen's Disease)

In 1876, Thomsen, a Schleswig physician, described a peculiar disease which up to that time had escaped observation. He had had experience of it in himself and numerous members of his own family. Thomsen called it "tonic convulsions of the voluntary muscles," an appropriate but somewhat clumsy name, for which we suggested instead the shorter name of "congenital myotonia."[1] Apparently the disease is very infrequent, but a considerable number of cases have already been reported in Germany, France, and Italy.

The disease is probably always congenital; at least, the symptoms invariably date from the earliest infancy. Occasionally, however, the symptoms are first noticeable approximately at the time of puberty. It is very often generic and hereditary; and males of the same family seem to suffer oftener, and also more severely, than do females. The essential symptom of myotonia is this: whenever any voluntary muscle has been inactive for a time and is then made to contract, it falls into a state of more or less persistent contraction, a mild sort of tetanus, so that it cannot be immediately relaxed. It is obvious that this would interfere with any series of motions, and make voluntary movement difficult. The patient is not paralyzed at all, but he has a feeling of great resistance to be overcome in performing any act. Quick and accurate motions are often out of the question, so that, for instance, the patient cannot perform military duty. It is noteworthy that the stiffness temporarily disappears after the patient has been moving his muscles for some time. On going upstairs the first steps are often very stiff and laborious, while succeeding ones grow easier and easier. Mental excitement invariably exerts a very unfavorable influence, exaggerating the stiffness of the muscles. Cold, likewise, has generally an unfavorable action.

Upon physical examination the observer is frequently struck by the extraordinary development of the muscles. The size of the extremities, in particular, almost deserves the term "genuine muscular hypertrophy," although the strength is not always proportionately great. Certain extremely interesting variations in the electrical excitability of the nerves and muscles are, however, very remarkable. These were first studied in detail by Erb, and given the name of "myotonic reaction." The motor nerves show quantitatively a normal faradic and galvanic excitability. All single shocks give short contractions, but on continued excitation (faradic current, labile galvanic current), a very characteristic persistence of the contraction occurs after the cessation of the irritation. The muscles are very easily excited by faradism, and, with rather strong currents, always show the above-mentioned persistence of the contraction. When the current is applied continuously, peculiar wave-like, oscillating muscular contractions occur. On galvanic excitation of the muscles a slight increase of excitability is manifest. With rather strong currents the contractions are decidedly slow, tonic, and also persistent. They occur only on closing the current, not on opening. Finally, we almost always see in the muscles a very remarkable phenomenon, discovered by Erb—rhythmical wave-like contractions occur in the muscles, under the action of a stabile current, which start from the cathode and go to the anode. Very characteristic, and of great practical importance (because readily demonstrable at any time), is the increased mechanical excitability of the muscles on tapping with the percussion hammer. After every blow (e. g., on the deltoid, biceps, etc.) the depression made in the muscles persists for a time. The so-called idiomuscular contractions are not increased. The reflexes, sensation, and, indeed, all other nervous phenomena, are normal.

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[1] [This is not to be confused with congenital myotonia.—K.]

These facts render is very probable that the cause of the disease is to be sought in the muscle itself, and that myotonia is due to a congenital peculiarity of the muscular system. This hypothesis is supported especially by the interesting histological discoveries of Erb on excised bits of muscle. He found a considerable increase in the volume of the individual fibers, whose transverse striation was often very fine and indistinct, at times the formation of vacuoles in the fibers, a great increase of nuclei, and a moderate increase of interstitial connective tissue. Nothing is known at present in regard to anatomical changes in the nervous system. Erb reports that, upon microscopic examination of minute particles of muscular tissue which were excised, he found marked hypertrophy of individual muscular fibers, and an increase in the number of nuclei in the sarcolemma.

The disease persists through life. The patient gradually becomes accustomed to it, and learns to conceal his misfortune as much as possible. There may be no constitutional disturbance. Sometimes there is melancholia. In a few cases the combination of myotonia and muscular atrophy has been observed. This form of myotonia has been designated as myotonia atrophica. Therapeutic efforts have not yet been attended with much success. We might try cold sponging, with friction, gentle massage, and methodical exercise of the muscles.

[Eulenburg describes a congenital family affection, which he calls congenital paramyotone, allied to myotonia, where tonic spasm was produced by cold. Gowers has found the association of tonic spasm with ataxia—ataxic paramyotone.—K.]

CHAPTER XI

HYSTERIA

ÆTIOLOGY AND DEFINITION

It is impossible to give a definition of hysteria that shall be at once brief and accurate, for the aspects of the disease are so manifold that there is no one symptom which can be called pathognomonic or even universally characteristic. Hysteria is therefore symptomatically not a well-rounded morbid unit, although the nature of all those pathological conditions which we term hysterical may very well be regarded from a single point of view. Hysteria thus has its peculiarities and its rules like any other disease. Only because its laws have been sought elsewhere than where they really are, has the claim often been made in the past that the morbid symptoms of hysteria were subject to no law.

The fundamental condition for a right understanding of hysteria seems to be that we should, on the one hand, free ourselves from the old untenable idea that hysteria is in any way connected with diseases of the sexual organs (*vide infra*); but, on the other hand also, that we should realize as well the incorrectness of that widely disseminated view according to which hysteria is a "general functional disease of the whole nervous system, where now this and now that portion of it may be disturbed in its functions." We regard hysteria as a disease which relates to the cerebral activity immediately associated with the psychical processes—that is, one may say, with a psychosis, but in the broader sense of the word; the disturbance affects not only the normal course of the psychical processes, but also their association with the purely corporeal processes of innervation. We therefore call any symptom hysterical which is due to a disturbance of the normal relations between the processes of our consciousness and our corporeity. Between our ideas of volition and our muscles, as between the excitations of our sensory nerves and our consciousness, there exist under normal conditions very definite and very well-ordered connections. If there comes a loosening, a displacement, a false estimate in these connections, it gives rise to a "nervous" disturbance of motion or sensation, an "hysterical" symptom; but the starting point of the disturbance always lies ultimately in the psychical domain. If, for example, the idea of volition suffers a morbid change in the sense that it can no longer arouse a corporeal motor excitation in any muscular territory, an "hysterical" paralysis follows. If the consciousness turns permanently from definite sensory impressions that come to it, we have to do with an hysterical anæsthesia. Later deductions will prove more plainly this characteristic psychogenetic origin of all hysterical symptoms.

In the meantime we may add to what has been said the following characteristic features of hysterical affections:

1. All hysterical disturbances, no matter how severe the functional nervous derangement attributable to them, are without visible anatomical basis. The best proof of this is the fact that any hysterical affection, however severe, may sometimes develop, and then may disappear completely in a very short time.

2. All hysterical affections are intimately associated with psychical events. Not only are their appearance and incipiency most closely linked with emotional excitement, but, later on, the mind is the main, if not the only, channel through which causes can operate to change the condition of the patient, whether favorably or unfavorably.

This peculiarity, which is deducible from the nature of hysteria, is at present generally designated as so-called "suggestibility" of the patients, and we speak in hysteria and allied affections of "suggestion," "autosuggestion," etc. These newly introduced foreign words have, however, the disadvantage of making simple and clear conditions appear unnecessarily complicated, and at times of imbuing them with a certain mystical air. At all events, it must be perfectly clear in our minds that the word "suggestion" does not mean anything else than the representation or imagination of some definite object elicited in any possible way, and that the word "suggestion" can always be entirely replaced by the above-mentioned terms. The definition given by Möbius, that hysteria is any disease which is produced by the imagination, is absolutely correct, but we must add that a primary conscious morbid representation is not always demonstrable in every case of hysteria. In psychopathically predisposed persons, hysterical symptoms occasionally appear apparently spontaneously, just the same as any other psychosis (melancholia, etc.) may develop "of its own accord." A morbid alteration of consciousness and the disturbance of the relations of ideation to physical conditions are, however, always the foundation and essence of all hysterical affections.

3. It is therefore evident that the origin of all hysterical disturbances must be sought in those parts of the brain which are the most central portions of the nervous system—those regions which are most directly concerned in the mental processes. Hysterical phenomena are, however, exhibited in all the various organs, so far as their functions depend upon psychical processes (i. e., certain mental representations) or may be altered by them. The symptoms of hysteria are therefore more manifold than those of almost any other disease. Although so manifold, certain symptoms predominate with such frequency as to be characteristic of hysteria, and therefore to be regarded as especially valuable in diagnosis. Following Charcot, we term such symptoms "hysterical stigmata." These symptoms are most constantly present, and therefore they can easily be found at any time.

4. Besides the permanent symptoms—the "stigmata"—we see in many cases of hysteria peculiar nervous attacks. These are also highly characteristic of hysteria, since they come on in forms which are seen in no other disease; but there are also patients who suffer from hysteria who nevertheless are never visited by attacks. The possibility of the occurrence of an attack is, however, always present. The hysterical attack is likewise almost invariably induced by special psychical factors. Frequently the psychical cause is readily demonstrable. Not infrequently, however, it eludes observation. After the attacks become frequent, only the slightest stimulus is sometimes required to produce a new attack.

[Babinski has lately urged that hysteria is merely a morbid suggestibility and that many of the symptoms, such as hemianæsthesia, contracted field of vision, etc., are merely artificial products due to suggestion. He suggests the word "pithiatism" instead of hysteria, and believes that only those symptoms should be regarded as hysterical which can be produced by suggestion and removed by persuasion. Babinski's position is somewhat too extreme, and cannot be fully accepted, although the influence of suggestion is very great. Janet and Prince lay chief stress upon the tendency to dissociation of the personality with mental depression and a limitation of the field of personal consciousness.—K.]

Turning to the special ætiology after this brief introduction, psychical causes, as has been said, are of first importance. In numerous instances hysteria comes on as an immediate sequel to violent emotional excitement or to a "psychical trauma," if we may use such an expression. Hysterical convulsions or paralysis may be excited by great terror, violent anger, or any unusual agitation. Here the efficient psychical cause is often hidden by some attendant circumstance. If, for example, a patient falls into the water, or gets burned, or tumbles downstairs, and thereupon develops hysteria, the mistake is often made of ascribing the subsequent nervous disease to catching cold, or to the injury received, although really it was the mental excitement which produced it. The various physical injuries (falls, blows, burns, electric shocks, etc.) play a far greater, and perhaps the greatest, part in the genesis of hysterical conditions. In such cases it is not the physical injury itself, but the terror it causes, or rather the whole disturbance of consciousness due to the trauma (terror, anxiety about the consequences, all sorts of fears, imagination, etc.), which produces the hysterical symptoms. These nervous affections produced by physical injury (traumatic hysteria, traumatic neuroses, neuroses due to accidents) are of such great practical importance that we will devote special consideration to them later.

What is very remarkable in such cases is that the special attendant circumstances of the psychical disturbance often influence the localization of the hysterical phenomena: that part to which attention was particularly directed at the time, not infrequently becomes, later on, the seat of the nervous disturbance. In hysterical joint affections (page 184) the cause not infrequently proves to have been an injury to the particular joint now suffering from hysteria. A young girl who was awakened at night by the smoke of her burning mattress, and who had a severe laryngitis from inhaling the vapors, exhibited later an indubitably hysterical paralysis of the vocal cords. In the case of another girl, who in jumping from a carriage fell upon her side, we afterwards saw hemianæsthesia of the same side. Many such instances could be cited.

Certain hysterical cases, therefore, have for their obvious cause a single occasion of mental agitation, but in many others the disease is not thus abruptly excited. As, in poisoning, we can distinguish between the sudden action of a large dose and chronic cases where minute amounts of poison are absorbed daily for a long period, so hysteria may come on not only after a single violent shock, but also as the final consequence of psychical influences insignificant in themselves, but potent because of their frequent repetition or persistency. It is in cases of this sort that the causes do not become evident to the physician until he has gained the entire confidence of his patient, for the root of the trouble is often entwined about the most private affairs. Anxiety, sorrow, disappointed expectations, abandoned hopes, and, in brief, everything which can depress and overwhelm the mind—these are factors which may at last excite the functional nervous derangements of hysteria.

All the ætiological factors enumerated so far, as a rule, however, constitute merely the exciting factors of hysteria. The real fundamental basis of the disease must be sought for, at least in a majority of the cases, in a congenital and hereditary abnormal predisposition of the nervous system. The blow which would bring a feeble body to the ground rebounds without effect from a massive frame. Exactly the same may be said of the "blows" to which the nervous system is subjected. Few entirely escape these influences, but there are some "strong natures" who resist the psychical assault without wavering, while others have a feebly resistant ("inferior") nervous organization and are overpowered. We see, therefore, that the predisposition of different individuals to diseases of the nervous system varies. This fact is a very important one in the pathogenesis of all functional nervous disorders. In what this predisposition consists we do not know, being acquainted only with its results and with some of its causes.

In many cases this predisposition is hereditary. Hysteria is most prominent among those neuroses which attack different members of a family—one suffering from one disease and another from another (*vide* page 582). It is also possible to acquire such a predisposition. At least it may be developed and fostered, on the one hand, or, on the other, checked and repressed. In these directions physical as well as psychical factors are of importance. Anything which weakens the constitution diminishes the resistant power of the nervous system. Among psychical influences, nothing favors the development of hysterical tendencies more efficiently than does a bad education. Hysteria is often ascribable to an irritability and feebleness of the nervous system thus engendered. The whims of the child are not controlled, its will is not strengthened or its energy developed; its imagination is unsuitably and excessively stimulated, or its intellectual powers are overtaxed and prematurely ripened.

It is well known that hysteria occurs more frequently in the "feebler" female sex than among men; but it is by no means a rare thing for men to exhibit hysterical convulsions, paralysis, contracture, or other well-marked disturbances. Most cases occur between puberty and the end of middle life. It is quite common to see pronounced hysteria in children, especially such as are over eight or ten years old. The disease very frequently begins to develop a year or two previous to puberty. Nationality and race also seem to exert some influence—for instance, the severer forms of hysteria are decidedly more frequent in France than in Germany, but severe hysteria is by no means rare in Germany. The Jewish race are particularly subject to hysteria, and to nervousness in general. The calling in life and the degree of intellectual development do not play a decisive part. Hysteria is found as frequently in the most intellectually gifted and cultured women as in the dullest peasant girl.

One matter remains to be considered with regard to ætiology, to which a greatly exaggerated importance was formerly ascribed. It is the influence of disease of the sexual organs. The very name "hysteria" (ὑστέρα = uterus) reveals what the old view was—namely, that hysteria invariably originated in disease of the female genitals. Not to speak of the fact that the disease occurs in men and children, an unprejudiced consideration of the matter will show that the above assumption is entirely groundless even in regard to women. A large number of hysterical women present no anomaly of their sexual organs; and even if the latter be diseased, we are not justified in assuming at once that the hysteria is secondary to the sexual disorder. In these cases, also, we usually find, on careful inquiry, that psychical causes have been at work, and these are incomparably more potent in exciting hysteria than is malposition of the uterus or constriction of the cervical canal. It is true, however, that disease of the genital organs may depress the spirits more than some other diseases would, and so indirectly promote hysterical disturbances. In the same indirect way, menstruation, pregnancy, and parturition exert an important influence upon the development and course of hysteria. Sexual excess or total abstinence from sexual indulgence also produce their effects indirectly through the mind. I regard as most unfortunate and utterly to be repudiated the opinion recently maintained by some physicians [Freud and others], that psychical sexual causes are always the basis of hysteria, and that it is the physician's task to ferret out these half-slumbering factors from the depths of consciousness, and to make them the basis of further "psychical" treatment. The extremely grave consequences of these views cannot, in our opinion, be combated with too much energy.

THE SYMPTOMS AND MANIFESTATIONS OF HYSTERIA

Considering the great differences in the external morbid symptoms under which hysteria may manifest itself, it is not easy to give in a brief summary a comprehensive, intelligible, and yet accurate description of its clinical manifestations. We believe that we can best attain this end by speaking first of the most important and most frequent of the single symptoms of hysteria, by describing next the hysterical attacks, and finally by trying to evolve a general picture of the disease.

The Hysterical Stigmata, Especially Sensory Anæsthesia and Hyperæsthesia.—As already mentioned, we designate as stigmata certain symptoms which are characteristic of hysteria, and the demonstration of which alone frequently makes the diagnosis of hysteria positive. A knowledge of these symptoms is the more important for the physician in that we often detect them only by a special examination directed to that end. Patients only exceptionally inform us as to these symptoms. It is not at all unusual for patients to have no inkling of their existence until after an examination. Indeed, it may even be maintained without hesitancy that numerous hysterical stigmata develop only through the medical examination, inasmuch as the ideas of the patients are influenced in an appropriate manner only by the examination. From this, it is evident that, from therapeutic considerations, a too extensive investigation into hysterical stigmata must not be made. The more certain hysterical patients are examined, the more are psychogenic symptoms at times developed. It is, therefore, a matter of medical experience and tact to determine to what extent each individual case of hysteria shall be examined.

The most important, because the commonest, of these hysterical signs relate to sensibility. The more carefully we examine, the more rarely will we meet with a case of severe hysteria in which in some sensory region we do not find manifest disturbances, especially a diminution of sensibility. We must therefore test accurately not only the sensibility of the whole surface of the body, but also the functions of all the special senses (sight, hearing, smell, and taste).

The sensory disturbances of the skin must first be mentioned. Not infrequently we find a diminution of sensibility over the whole surface of the body, especially a more or less complete analgesia. In such cases we can stick a pin in deeply anywhere, or pierce a fold of skin with a pin, without the patient's complaining of pain. As is well known, it often happens that hysterical patients, in order to appear interesting or from some other reason, produce upon themselves deep injuries and wounds; this is almost always connected with the patient's analgesia. Very often, however, the anæsthesia is not general, but is limited to a definite region. We find complete anæsthesia of one arm or leg, or we find on the trunk or the extremities single areas of anæsthesia, in which the anæsthetic parts are bounded by normally sensitive skin; these areas may take all possible forms, and may often be very peculiar. The sensibility to pain is almost always the most disturbed, even when the other forms of cutaneous sensibility may be quite unaffected, but changes in the sensibility to temperature, pressure, etc., may also occur. It may be added that in the severer forms of anæsthesia the deeper parts may also be insensitive, so that the so-called muscular sense is absent, and patients with their eyes shut have not the slightest idea of the position of the anæsthetic limbs.

Sensory disturbances of the mucous membranes are next in order. Here the hypæsthesia is noticed particularly frequently in the conjunctiva and pharynx. Hysterical patients can, as a rule, bear a touch on the conjunctiva without winking, and can have their pharynx tickled without getting a gagging reflex. This absence of the pharyngeal reflex is a particularly common and, practically, a not unimportant stigma of the hysterical constitution. [The skin reflexes are often diminished or absent over the anæsthetic regions. The tendon reflexes are often exaggerated, but occasionally diminished, on the affected side in cases of hysterical hemianæsthesia and hemiplegia. It is very doubtful if loss of the knee jerk, true ankle clonus, or a Babinski reflex ever occurs in uncomplicated hysteria.—K.]

Besides cutaneous anæsthesia, disturbances in the sensibility of the other sensory organs (other " sensory anæsthesias ") are among the commonest signs of hysteria. Sometimes the patients say, when asked, that they see indistinctly and dimly. If we examine the eyes we often find a loss of visual acuteness and a rapidly induced fatigue on using them. A limitation of the visual field is the most characteristic sign—that is, an anæsthesia of the peripheral portions of the retina. The degree of this limitation sometimes shows manifest differences for the different colors. Charcot devoted particular attention to the hysterical achromatopsia—i. e., the loss of color sense. According to Charcot, in the hysterical the perception of violet usually first disappears, then green, and, lastly, blue and yellow; but we must not hold too strictly to all these rules, for, although the French investigators are otherwise deserving of great praise in adding to our knowledge of hysteria, in this respect they seem decidedly too schematic, and to have generalized too rashly. Diminution of the auditory acuteness in one or both ears is also not uncommon. Anæsthesia of smell and taste is still more common. Salt, quinin, vinegar, sugar, etc., either excite no sensation of taste at all, or the tongue is anæsthetic for one or another of these substances. The same holds for the sense of smell. All these symptoms are especially important in diagnosis, because in organic diseases they seldom reach such a degree as in hysteria.

The sensory anæsthesias above described may, of course, be combined in various ways in different cases, but none of these combinations is so characteristic and individual as hysterical hemianæsthesia, a symptom-complex which occurs almost solely in hysteria, and is therefore of distinct diagnostic value.

Hysterical hemianæsthesia is one of the most common symptoms of profound hysteria. It must often be sought for, inasmuch as the patient herself frequently has no suspicion of its existence until her attention is called to it. It seems just as if one half of the body had been entirely lost to consciousness; the patient does not know whether it is or is not capable of feeling.

In a typical and fully developed case the hemianæsthesia does actually affect just one half of the body. There are rudimentary forms, but in these complete ones, the boundary between the anæsthetic parts and the parts retaining normal sensitiveness accurately corresponds to the median line of the body. The skin on the affected side is entirely insensible to the prick of a needle or to heat. Often it seems somewhat blanched, and its blood vessels seem to be constricted; at least the skin very often bleeds surprisingly little if wounded. The mucous membranes upon the abnormal side, including the conjunctiva, and the corresponding half of the buccal cavity and of the tongue, are all equally anæsthetic. The deeper parts, such as the muscles and joints, are also almost invariably anæsthetic. The patient can no longer feel in what position the limbs of the affected side are; and, if they are moved passively, no sensation is communicated to her. The organs of special sense are usually involved. Hearing is impaired upon the affected side, the corresponding half of the tongue cannot taste, the corresponding nostril cannot smell, and sight upon that side is affected in a peculiar manner. There is no hemianopsia, but a total amblyopia, or possibly amaurosis. If the amaurosis be not complete, we find at least a limitation of the visual field and the signs of the above-mentioned achromatopsia.

Apart from other hysterical symptoms with which hemianæsthesia as well as all the other sensory anæsthesias may be associated, we must mention here one symptom which is in close relation to anæsthesia, and which was first described by Duchenne under the name of "loss of muscular sense" (*perte de la conscience musculaire*). The patient, whose arm, for example, is anæsthetic, cannot move it if she closes her eyes, although with the eyes open she can move it as well as ever. When the eyes are shut the arm remains motionless in the position it has previously occupied. If its position be altered by passive motion, the new position is maintained with equal persistency. There is, with the eyes shut, pronounced catalepsy. Duchenne referred this peculiar symptom to the loss of a special sense which he termed "muscular sense" (*conscience musculaire*). According to our present conception of hysteria we must undoubtedly regard this symptom as purely psychical.

[The various anæsthesias and many other symptoms of hysteria might often be termed more properly "delusions of not-feeling." Patients with hysterical blindness, for example, when prisms are placed before their eyes, so that they imagine they are looking with the sound eye when they are really looking with the "blind" eye, can sometimes see very well. When hypnotized such patients can recall that they have been touched or pricked in the anæsthetic area, etc. This is often mistaken for deliberate simulation, but it is really another instance of the psychical nature of the disease.—K.]

The much-discussed and peculiar symptoms of transfer, as well as metalloscopy and allied symptoms, will be mentioned below.

Thus far we have spoken only of anæsthesia, but we very often find in the hysterical certain hyperæsthetic regions. These hyperæsthesias are so characteristic that we must reckon them among the hysterical stigmata, and must therefore look for them in every case. Sometimes the physician's attention is called to these places because they are the seat of constant slight or severe pain. In other cases the tenderness appears only upon pressure. The hyperæsthesia may be so great that the patient can scarcely bear the slightest touch. Of course, the hyperæsthesia is very closely connected with the condition of the patient's attention. If the thoughts be diverted, even firm pressure is often not noticed at all. We see clearly, then, that we usually have to do here with a "psychical hyperæsthesia."

The hyperæsthetic spots are either quite extensive or quite circumscribed, or even limited to one definite spot. They may lie near to or in the midst of anæsthetic areas. They are relatively rarest in the extremities and more common on the head and trunk, especially on the sternum, on the sides of the chest, under the mammæ, etc. Most common, and therefore especially characteristic, is the hyperæsthesia of the vertebral column, and, in women, of the lower abdominal region ("ovarian pain," "*ovarie*"). The latter term is convenient, but by no means correct, since really we have only a tenderness of the soft parts in general, which is at once noticeable on deep pressure, and we do not have solely or chiefly a tenderness of the ovary. We usually find *ovarie* only on one side, oftener on the left than on the right. Analogous symptoms in men—tenderness in the groins or the testicles—are less common. Hyperæsthesia of the vertebral column, hysterical "spinal irritation," is even more important for diagnosis than *ovarie*. The hyperæsthesia affects either the whole spine or merely single portions of it, often only single vertebræ. It may attain so high a degree that the slightest touch of the skin over the vertebræ, or somewhat deep pressure, may cause the liveliest outcries and shrinking. This symptom is almost pathognomonic of hysteria.

The relations which these hyperæsthetic regions have to the hysterical attacks are very important, and they have therefore procured for them the name of "hysterogenous zones." We will speak further on this point in describing the hysterical attacks.

Hyperæsthesia in other sensory regions—abnormal sensitiveness of the eye, ear, etc.—also occurs. Thus, some cases of hysterical blepharospasm seem to be due to an immoderate sensitiveness to light. Unusually sharp hearing is noted, especially during the hysterical attacks. Here, too, belongs the aversion of many patients to certain odors and flavors and the like.

We may also mention in this place hysterical pains. They can scarcely be separated strictly from hysterical hyperæsthesia; at least, almost all hysterical patients, who complain of pain (in the back, in the face, in the arms and legs, and very often "all over"!), state that these pains are increased by motion and pressure. The more the patient's general disposition is disturbed, the more he thinks of his pain and the worse it becomes. If the patient is busied with other things he forgets his pain entirely. Suggestive influences (*vide infra*) are of great effect. There can therefore be no doubt that these feelings of pain are of a purely psychical nature; they are, so to speak, "imaginary pains," hallucinations of pain, or, if we please, "central pains"—i. e., central psychical excitations, which are manifested subjectively as pains. The hysterical nature of the pains can frequently be recognized by the experienced physician by the associated general psychical excitement and ætiology, by the exaggerated manifestations of pain and similar circumstances. A patient with true appendicitis, for example, lies as quietly as possible in order not to increase the pain, whereas the patient with hysterical enteralgia tosses restlessly in bed. This restlessness, however, is not found in all hysterical subjects. Hysterical pains sometimes become very important because they render the patient inactive. There are hysterical patients who lie the whole day on the sofa, because they cannot stir a limb "on account of pain." Möbius and others have described such especially pronounced cases under the name of "*akinesia algera.*" In our opinion, there is no reason for wishing to make a separate disease of such cases, because such conditions often enough occur alone or associated in various ways with other symptoms in severe neurasthenia and hysteria.

Hysterical Paralysis.—Hysterical paralysis is frequently an immediate sequel to some violent mental excitement (for example, "paralysis from fright"), but it may come on gradually. Hysterical paralysis is indubitably of central origin. It is a paralysis of the will. The patient has lost the power to will a contraction of the affected muscles. One always has the feeling that the patient could move the paralyzed limb perfectly well if she only desired to, but she cannot bring the will to bear on it, and this inability is the real trouble. It also seems a remarkable circumstance that hysterical paralysis often, but of course not always, affects only certain combined movements. Many patients, for instance, can move their legs very well in bed, but they cannot walk a step ("*abasia,*" *vide infra*). We have seen an hysterical writing paralysis; the right arm was not paralyzed, but it utterly refused to make any attempt to write.

The extremities are most frequently paralyzed, particularly the lower limbs; but hemiplegia is not especially rare. A very common manifestation is loss of the power to stand or walk. The patient lies in bed or on a sofa, and sometimes while thus reclining she can flex and extend the legs very well; but as soon as she is urged to stand up or walk, the knees double up, the patient begins to tremble, the respiration grows rapid and convulsive, and there is not the slightest effort made to move the legs. In France the names "*astasia*" and "*abasia*" are used for these common hysterical symptoms. If only one leg be paralyzed, the gait is often very peculiar and characteristic. The sound limb makes long strides, while the paralyzed one is held perfectly rigid, and often is dragged along with a loud shuffling sound. The arms are much less often affected. The facial muscles are hardly ever paralyzed, although they may be involved in hemiplegia. We once saw hysterical diplopia.

In regard to the more precise form of the paralysis, both flaccid and spastic paralyses occur in hysteria. In many cases of hysterical paralysis the tendon reflexes may be so exaggerated—nay, there may even be, as we have repeatedly seen, such a lively ankle clonus—that one is at first disposed to think of actual spinal paralysis. In other cases, as in paralysis of an arm, the paralyzed limb hangs down completely flaccid. The paralyzed parts are often at the same time completely anæsthetic (a circumstance which may often be distinctive in diagnosis), but in other cases they are normally sensitive or even hyperæsthetic.

Hysterical paralysis of the vocal cords is very often seen. The voice is generally lost suddenly, and the patient can talk only in a whisper—hysterical aphonia. On laryngoscopic examination, we are often struck, at the outset, by the anæsthesia of the pharynx and its lack of reflex excitability. We find no trace of any anatomical lesion of the cords, but merely that they are paretic. The glottis cannot be completely closed, and sometimes the vocal cords actually become wider apart upon every effort at phonation. The patient then speaks exclusively in a whisper. Hysterical mutism quite rarely follows hysterical aphonia. The patients entirely lose voluntary control of their speech apparatus, and finally become completely dumb. We will also mention here hysterical stammering, although it should more properly be classed with the spasmodic hysterical symptoms. The voluntary quiet innervation of the muscles of articulation is affected, so that the speech is altered in the most peculiar way, by needless spasmodic attendant movements, grimaces, etc. We have very often observed such hysterical disturbances of speech (stammering or mutism), especially in children (as a result of fright). A loss of the power of writing may sometimes be associated with the dumbness in an interesting way.

Hysterical paralysis of deglutition is much less frequent than paralysis of the vocal cords. If there be dysphagia, it is often not an easy matter to determine whether it is due to paralysis or to spasm which comes on at any attempt to swallow.

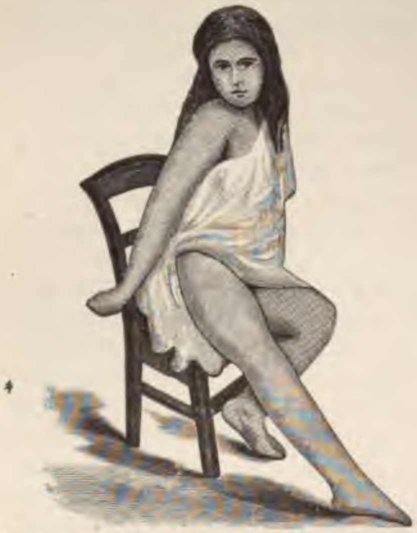

FIG. 219.—Hysterical contracture (from BOURNEVILLE and REGNARD'S "Iconographie de la Salpêtrière").

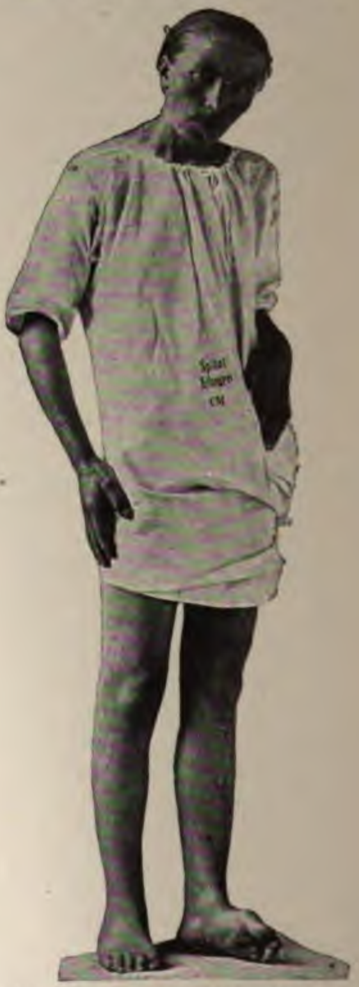

FIG. 220.—Contracted position of the left foot in traumatic hysteria (personal observation).

Hysterical Contractures.—Contractures may occur as isolated phenomena or in combination with other symptoms, such as anæsthesia or paralysis. They are caused by unusually strong tonic muscular contractions. The starting point of the irritation is certainly in the central nervous system. Sometimes the contractures are temporary, but often they are characterized by their intensity and their great persistency. The extremities suffer most frequently, although the trunk or the back of the neck may be affected. In the hands and feet, especially in the toes, flexor contractures predominate, but in the larger joints extensor contractures are the rule. Although many varieties occur, some forms of contracture are especially characteristic. As an example, we will refer to the accompanying illustrations (Figs. 219, 220).

The relations of hysterical contractures to the articular neuralgias have already been mentioned (page 184). They may occur in hemiplegic, paraplegic, or monoplegic form. They often follow an hysterical convulsive attack (*vide infra*). All hysterical contractures disappear completely in chloroform narcosis, after the use of an Esmarch bandage, and in deep sleep.

Vasomotor Disturbances. Secretory Disturbances. Symptoms in the Internal Organs.—Besides the sensory and motor symptoms just described, other symptoms occur in hysteria which fall under the domain of the vasomotor and secretory nerves; but in our opinion we should cite here with positiveness only those symptoms which we find to be dependent directly or indirectly upon psychical processes. In this domain, however, the credulity of many physicians has brought to light a mass of apparently wonderful and inexplicable observations, which are not proof against rigid criticism.

In the first place, abnormal anæmia or abnormal fullness of the vessels (a cool, pale skin in the one, a hot, red skin in the other) is not infrequent, and points to vasomotor influences. Since we know that the vascular nerves are influenced in a high degree by the emotions, we can probably assume a central origin for these symptoms. Hemorrhages from internal organs are harder to explain; they are apparently not uncommon in hysteria, and they have often been referred to "nervous vasomotor influences," but in our opinion we must be extremely guarded in such an assumption. We most frequently see hysterical hematemesis or hysterical hemoptysis—that is, the evacuation of blood from the mouth, either with cough or in a way suggestive of choking. Inexperienced physicians have thus often been led to a false diagnosis of dangerous pulmonary disease or gastric ulcer; but if we look more closely we shall find that the blood evacuated is of a clear raspberry-red color, is mixed with a good deal of mucus and saliva, and is of a thin fluid consistency. The whole amount is seldom more than 2 or 3 ounces (50 to 80 c.c.). If we inquire more carefully as to its origin, we can usually find a source for the blood in the gums or in the oral or pharyngeal mucous membrane. There are often spasmodic chokings or hiccoughs, by which a hemorrhage of the mucous membrane is mechanically produced. It is also certain that in many cases intentional deception on the part of the patients may lie at the bottom of the hemorrhage. We must be extremely cautious in hemorrhages from the genitals, from the skin, from the palms in the "stigmatized," etc. We once exposed an hysterical woman who had for years produced the most extensive gangrenous spots upon her skin by caustic soda!

Finally, among the vasomotor disturbances we must mention one symptom whose significance, in our opinion, has not yet been satisfactorily explained—we mean hysterical fever. In severe hysteria, especially at the time of severe attacks and psychical disturbances, other observers, and we ourselves, have repeatedly seen high febrile temperatures, 106° F. (41° C.) and more; and these temperatures come on in a very irregular manner. In none of the cases under our own observation, however, could we wholly exclude the possibility of simulation—that is, of producing a rise of the mercury by rubbing and pressing on the thermometer. In all the measurements we made ourselves in the rectum, the temperature was normal, while the high fever was always said to occur in our absence. We therefore recommend the greatest caution with reference to this point, although we will admit that a rise of temperature may occur from purely nervous causes.

Anomalies of the secretory and excretory organs have also been met with in hysteria. Many patients have a remarkably dry skin, while others sometimes perspire very freely. The secretion of saliva is subject to similar modifications. Very remarkable observations have been made in a few cases with regard to hysterical ischuria; for days only a very small amount of urine has been passed, although there has been no retention. In one case of this sort, observed by Charcot, there was violent vomiting at the same time, and the vomitus was said to contain a comparatively large amount of urea (vicarious excretion). Such observations, which are very few in number, are at present not susceptible of an exact explanation, but the suspicion of simulation does not seem to us wholly excluded. Hysterical polyuria occurs more frequently than ischuria. A large amount of very light-colored urine of low specific gravity is excreted. This polyuria is in many instances merely the result of excessive ingestion of liquids. Polydipsia (excessive thirst, or probably, to speak more correctly, a morbid tendency constantly to drink water) is a very frequent symptom in hysteria, particularly at the close of an hysterical attack.

The digestive disturbances which many patients have are mainly such as have already been discussed (*vide* Vol. I) under the head of "nervous dyspepsia." Colicky pains, obstinate constipation, occasional diarrhea, and similar symptoms, are by no means rare. Hysterical vomiting, especially, often plays a very great part, since it may very much impair the patient's nutrition. In other cases, of course, the patient's well-nourished appearance is in striking contrast with her statement that she "has to vomit everything up again." Hysterical tympanites also deserves mention. It is due to the accumulation of a large amount of air and gas in the intestines. This may be in part the result of a sort of paralysis ("atony") of the muscular coat of the stomach and intestines, but another frequent cause is the swallowing of large amounts of air. Perhaps the patient does this on purpose. The prominence and tension of the abdominal walls may be so considerable as to simulate grave diseases, such as peritonitis, a tumor, or pregnancy. Doubts of this sort can always be dispelled by inducing anæsthesia with chloroform. It is possible to remove the gas completely in a short time by pressing upon the abdomen, or by introducing a long tube through the rectum.

Sometimes there are symptoms referable to the genital organs. It has been already pointed out that too much prominence was formerly given to the influence of sexual diseases in exciting hysteria. It is also true that nervous derangements of the genital organs, as of all the other organs, may be among the symptoms of hysteria. Many pains and hyperæsthetic conditions of the genital organs must in particular be so interpreted, while coexisting disturbances of menstruation are often to be regarded as coördinate symptoms. We can also readily understand that sexual relations often influence very excitable, hysterical individuals to no slight extent, as is indeed very frequently betrayed by the character of their hallucinations and their utterances when delirious.

General Mental and Bodily Constitution of the Hysterical.—If we regard hysteria as a disease which is essentially psychical, which is the only correct view, it will not seem strange if the whole mental constitution and nature of the patient show special peculiarities. In many cases, therefore, the psychical condition of the hysterical is so characteristic that the physician can, from the patient's nature and demeanor, form his conclusions as to the form of her disease.

Hysterical persons are irritable and emotional, easily depressed, sensitive, whimsical, and subject to violent extremes of feeling. They are inclined to exaggerate their sufferings, exact a great deal of attention, and are anxious to excite sympathy. They have little energy or force of will, but they are sly and obstinate in carrying out any pet desire. Again, they can be very amiable and attractive if they take the fancy. They are often shrewd and active mentally, but hysteria is also not infrequently seen in dull and stupid persons.

This brief sketch represents many cases, as we have said, but not all. The patients to whom it refers, as a rule, present no very great disturbances, but complain merely of all sorts of general derangements, sometimes of one kind and sometimes of another, and yet are able to perform their daily duties tolerably well. A case of paralysis, contracture, or other important localized hysterical trouble may not present any marked mental peculiarities. Often, of course, the patient conceals them from the physician, and the psychopathic nature of the whole mental constitution ("character") is finally revealed only by a closer acquaintance with the patient and her previous life.

With regard to the general physical constitution of hysterical subjects, it has already been mentioned that any bodily weakness favors the development of the disease; and yet hysteria is by no means confined to the ill-nourished, weakly, and anæmic. On the contrary, many patients seem to be in blooming health and well nourished. In a severe case of hysteria, however, the effects of the disease on general nutrition may be very distinct. Little food is ingested, sleep is disturbed, digestion is affected (*vide infra*), and the bodily health is gradually undermined.

HYSTERICAL ATTACKS, CONDITIONS OF SPASM, ETC.

While the symptoms thus far described are largely of a permanent character, paroxysmal nervous symptoms are also common in hysteria. The diagnosis of the entire morbid condition is often made certain by the onset of such hysterical attacks, which are often very characteristic, and their significance is readily perceived by the experienced physician. There are some cases of hysteria which run their course entirely free from these attacks, and present only certain permanent nervous symptoms, such as paralysis, contractures, anæsthesia, etc.; but there are also other patients in whom the hysterical attacks dominate the whole scene, and are sometimes the only manifestations of the disease. An hysterical attack is not infrequently the beginning of the trouble, especially in those cases where the disease is excited by fright, etc., and where the attack follows immediately upon the psychical excitement.

In regard to the severity and the variety of the hysterical attacks, they are so manifold that an exhaustive account of all the possibilities cannot here be given; but certain features and details are so common in them, and recur so often, that a consideration of them is often in itself sufficient for a correct diagnosis.

The mildest form of the hysterical attack consists of the development of a feeling of distress, anxiety, vertigo, and especially of a loss of voluntary control of the body. The patient sinks on a bed or chair, closes the eyes, and becomes incapable of action or speech. Mild symptoms of motor irritation usually ensue, most commonly an acceleration of respiration, general tremor, winking of the eyes, etc. Mild spastic symptoms in the pharyngeal muscles and the diaphragm are not uncommon. There is very often a lively palpitation during the attack. The face is sometimes quite red, but in other cases pale.

In such mild attacks any impartial observer has decidedly the impression that the patient is humoring her inclinations. If, therefore, she is cheered up, or if cold water be thrown in the face or on the back without too great regard for her feelings, she usually soon regains her will power and rapidly recovers.

In an unbroken series these mildest forms of attack pass over into the severer types, where the clouding of consciousness is greater and the symptoms of motor irritation are more severe. Complete loss of consciousness, so common in epilepsy, is never seen in hysterical seizures, but there is very often a marked clouding of consciousness, and in the severe forms morbid alterations of consciousness (*vide infra*). If hysterical attacks occur which resemble those of epilepsy ("hystero-epilepsy"), we must think of the possibility of a combination of the two diseases; but usually the form of the hysterical convulsion is essentially different from that of the epileptic. In severe hysterical convulsions the convulsive movements are more varied, more extensive, and more complicated than in epilepsy. The arms make thrashing and thrusting movements, and often apparently quite well-coördinated movements. Patients strike the bed, and sometimes their own bodies, with clenched fists; they seize objects, such as the bedclothes or furniture, and cling fast to them. We also see clonic or tonic flexor and extensor spasms in the legs. The eyes are almost always rolled up; more rarely they converge or are turned to one side, and they often roll about. The lids are tightly closed or occasionally open. There is almost always trismus, and there is often grinding of the teeth. The trunk usually twists and turns the most of any part of the body. The patient often hits her head violently against the wall or the bed. The whole body may assume positions which a healthy person can scarcely imitate without special practice. The best known, and in fact one of the commonest and most characteristic, is the arched position (*arc de cercle*), of which the accompanying illustration (Fig. 221) gives an idea. Many patients rest for a time on the floor supported only by the head and toes. At intervals they fling or roll their bodies to and fro, drum on the floor with their legs, throw themselves into the air, etc. Anyone who has once seen such a "grand hysterical convulsion" will never forget the picture.

A different but yet a common form of hysterical convulsion is characterized by a pronounced implication of the respiratory muscles. The attack begins with a spasmodic acceleration of respiration, and the breathing becomes progressively shorter and more rapid. We have ourselves counted two hundred respirations a minute. Other peculiar spasms of the respiratory muscles, which occur in this way only in hysteria, are also common—hiccough, loud sobbing, grunting, etc. The muscles of the pharynx are usually implicated in producing these noises. Of course, all these spasmodic conditions may be joined in the most varied ways with spasms of the trunk and the extremities. We very often find during the hysterical attacks pronounced cataleptic states. If the patient lies in bed with the eyes closed and rigid extended extremities,

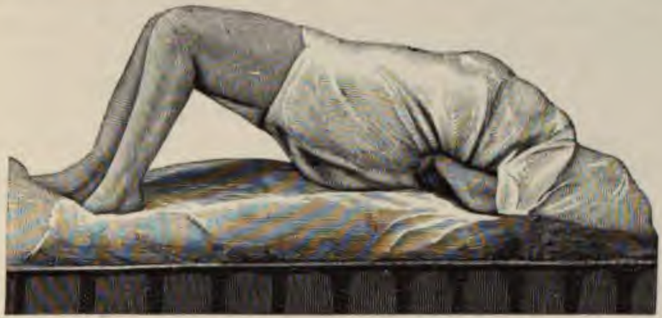

FIG. 221.—Hysterical *arc de cercle* (BOURNEVILLE and REGNARD).

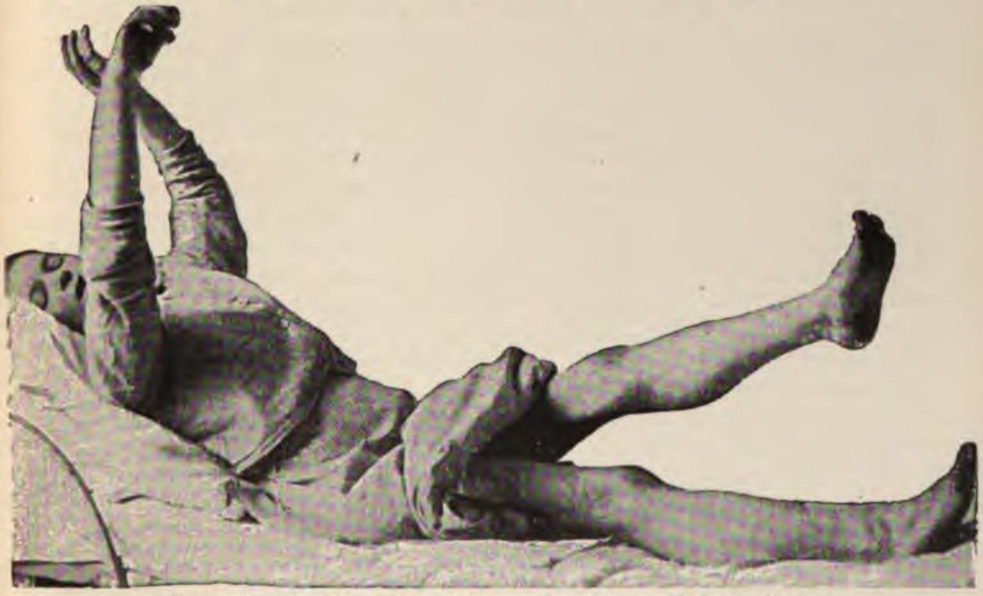

FIG. 222.—Hysterical attack with catalepsy (personal observation).

we can put the arms and legs into the strangest positions passively, and they are kept there for a long time (see Fig. 222). In other cases the cataleptic condition sets in only if the eyes of the patients, which were previously closed in the lethargic state, are forcibly opened. Charcot has described a few show cases of grand hysteria, in which he placed the patients in certain postures intimately associated with some mental picture (attitude of prayer, crucifixion, of fright, disgust, and others). The appropriate facial expression immediately ensued in these patients, and the appropriate position of the entire body was reproduced with the greatest theatrical realism. We see from this to what degree of suggestibility hysterical subjects (I might almost say) can be trained.

One of the essential factors, which often gives the most characteristic stamp to the grand hysterical attack, is the relation which, on the whole, the spasms often have to certain coexisting abnormal conditions of consciousness. During the attack the patient is usually not unconscious, but she is under the dominion of morbid delusions, and these often mirror themselves in the outward movements. The patient is wholly dominated by a definite circle of ideas; she has hallucinations, and passes through some frightful or exciting event. All this is most strikingly manifested in her movements and her expression. Hence, in an attack the face often expresses terror, rage, threatening, concupiscence, serenity, etc. Very often the internal excitement breaks forth in words, and there is actual hysterical delirium, the most marked emotional utterances, etc. We often observe, and it is a fact of especial interest, that in hysterical attacks which have come on immediately after a severe fright, or some exciting event, the same scene to some degree is rehearsed over in the patient's consciousness in each subsequent attack. If, for example, hysterical attacks come on in a nervous child who has been attacked by a dog, we can quite frequently recognize again the cries of terror at the approach of the dog in each new attack, etc. The patients often talk continually to themselves during the attack, usually very rapidly, with frequent repetitions of the same phrase or word. We can often succeed in giving the delirium a definite direction by questioning the patient, or converse regularly with the patient during an attack, but usually the pure motor spasms again set in, or persist for some time in a tonic form.

We cannot go further into the numerous details of the symptoms of the grand attack ("*grande hysterie*"), for an accurate knowledge of which we must thank the observations of Charcot and the school of La Salpêtrière in Paris (Bourneville and Regnard, P. Richer). The descriptions of the French authors apply in general to the grand hysteria occasionally seen in Germany. There exists, however, no doubt in our mind that a large part of the peculiar symptoms described by Charcot were artificially cultivated. The hysterical cases were for a time the "*enfants gâtés*" of the Salpêtrière. Charcot took a special interest in them. Little wonder, then, that suggestion and autosuggestion (*vide infra*) bore good fruit. The rules and laws laid down at that time require to be greatly qualified. The French neurologists distinguish several periods in the grand hysterical attack. The first period consists of severe epileptiform convulsions apparently associated with loss of consciousness. Then comes the period of contortions and grand movements (clownismus), and finally the period of plastic positions and passionate attitudes (*attitudes passionelles*). The short and very significant expressions of this scheme are of practical value, but according to our experience one can only rarely expect sharply defined "periods" in the individual case; the grand hysterical attack is rather composed in the most varied ways of the different symptoms above mentioned.

There remains one essential trait needed to complete the picture of the hysterical attack, and that is the "suggestibility" of the patient, which is so peculiarly characteristic of hysteria. By "suggestion" we mean the artificial production of a definite psychical state, or a physical state dependent on the mind, by arousing the appropriate ideas. In the general characteristics of the mental constitution of many hysterical persons we have already had to point out how much and how often such patients let themselves be dominated by their imaginations. Suggestion is merely the artificial fostering of this psychical peculiarity and its special application. The more we try to foster and preserve this peculiarity, the more we leave uncorrected the false ideas which the patients have, the better we succeed at last in making the patients merely a shuttlecock for their ideas. Hence the physician's daily experience teaches that, as suggestion experiments are repeated, the patients more easily become susceptible to them, so that finally we can actually "do everything with them." There can scarcely be any difference of opinion as to whether such an experiment is entirely justifiable, medically and morally; and even the fostering of suggestion for therapeutic purposes is a two-edged sword whose action is not always rigidly within the physician's control.

Suggestion is most easy during the hysterical attack itself, especially in those forms where the patients speak, hear, and answer. As soon as we give the patient's ideas a definite direction, and tell them in a convincing tone that they are in a garden or a wood, picking flowers or fruit, that they are attacked, bound, lying on the edge of a precipice or the water, etc., we see in their bearing and speech that they think they are actually experiencing all these conditions in their delirium and hallucinations. The emotional utterances of fear, terror, joy, or aversion are then often expressed with astonishing art. In the same way we can suggest paralyses, contractures, or anæsthesias. The most interesting feature of it all is, that, when the attack is over, every trace of recollection of what happened during it is lost. The same patients who just now were so much excited by some definite idea, a few seconds later, as soon as the attack has ceased of itself or has artificially been brought to an end (*vide infra*), know nothing more about it. They have not even a vanishing recollection like that of a dream about what they have just said and done, even if it be expressly described; but it is still more remarkable that during the following attack they often remember very well what they have experienced, actually or in their imagination, in the past attack. In such cases we can truly speak of "double consciousness." Processes of the conscious waking life, however, often remain in the consciousness during the attack. We have already said that the actual event (scene of terror, etc.) which has given rise to the first onset of the attacks is often revealed anew in the delirium of subsequent attacks.

Hypnotic symptoms are closely allied to the processes in suggestion. We cannot give here a complete review of this field, which, as is well known, has been so much studied of late. The tendency of human nature to mysticism and the influence of suggestions, which may be potent not only with the patients but even with the investigating physicians, are the reasons why the false and the true have often been mixed in the study of hypnosis. We can say only that one great advance is now generally recognized, and that is, that most persons of scientific training have abandoned the hypothesis, once frequently accepted, of a special "magnetic power" (animal magnetism), by which the "magnetizers" could put their "mediums" into the "magnetic sleep" or other abnormal conditions.

Hypnosis is nothing more than the intentional artificial production of an hysterical attack, or an hysterical psychosis by suggestion—that is, by the action of definite ideas on the person to be hypnotized. Therefore, only those persons can be hypnotized in whom these ideas have a strong enough influence. No man can be hypnotized to whom the nature of hypnosis is clear. The essential feature of all hypnotic procedures is merely to produce in the most lively way possible the idea, "It will happen as the hypnotizer says." All other things —the fixation of the eyes on bright objects, the vibrations of a tuning fork, etc.—are side issues, and serve merely to support the suggestion. In all easily hypnotizable persons the mere closure of the eyes and the remark, "Now go to sleep," are enough to produce the hypnotic sleep. Patients (and here we may actually speak of "patients") reach this suggestibility, of course, only after they have often been hypnotized; for the oftener the same action of an idea is produced the more easily it occurs—a law which follows also from many other experiments in the psychical domain. The different forms of hypnosis are not distinguishable from the various hysterical conditions. Hypnosis is an artificial hysterical symptom, designedly produced, and from this alone we note the danger of all hypnotic experiments if they are practiced by the ignorant. In this, too, it has often been shown that we cannot become free again from the spirits which we call up.

As a rule, we distinguish four chief forms of the hypnotic state, which show, however, many transitions: (1) The cataleptic state, in which the limbs retain all the positions artificially given them (see the previous chapter). (2) The state of "suggestion," of artificially produced hallucinations. If we put the body passively into certain positions corresponding to definite acts, we can produce in the patient all the appropriate ideas with the directness of an hallucination. To this category belong the well-known hypnotic exhibitions where hypnotized adult men dance dolls, eat raw potatoes with an expression of enjoyment, etc. (3) The lethargic state; that is, a state of apparent unconsciousness, with the eyes closed, the muscles completely relaxed, and a marked increase of excitability in the muscles and nerves. A light pressure or a slight blow on a nerve, such as the facial, suffices to put all the muscles supplied by it into a tetanic contraction which outlasts the irritation. (4) By certain manipulations (for instance, rubbing over the vertex) we can change the lethargic state into one of hysterical somnambulism. The patients remain half unconscious, but answer automatically questions which are put to them, obey orders given to them, and sometimes show certain sensory hyperæsthesias. We see that all these forms are precisely identical with the different forms of the hysterical attack. Only the increased mechanical excitability of muscles and nerves is not yet fully explained, but the reported observations upon this point have not yet been absolutely confirmed. It is especially a question whether suggestion—that is, ideas which lead to unconscious muscular contractions—may not also play a part here?

Another important point in the hysterical attacks must be discussed, the "hysterogenous zones." We have mentioned above how often in hysterical patients certain parts of the body (the ovarian region, the sides of the chest, etc.) are extremely sensitive to pressure. It is by no means uncommon that, in spite of the patient's resistance, somewhat longer pressure on such a spot may excite an hysterical attack. On the other hand, we sometimes succeed in causing an existing attack to cease by pressure on the same zone. We are of the opinion that in these manipulations also ideas become potent as connecting links; for we may, as I have demonstrated in my clinic almost every term, by suggestive persuasion or by all possible procedures excite and then inhibit attacks in hysterical patients. In such cases we never have to do with physiological reflex processes, but always with the action of ideas suggested to the patient.

Finally, we must add that there are also milder forms of hysterical spasm which are limited to a definite muscular region, and are not associated with any marked clouding of consciousness. There are, for example, isolated spasms of the muscles of the neck, isolated respiratory spasms (spasmodic cough, etc.), not infrequently associated with the strangest noises, and isolated spasms in the arms and legs; the laryngeal muscles may also be affected (hysterical spasm of the glottis). Spasms of the diaphragm and other muscles of inspiration are quite common under the form of hysterical hiccough, which may sometimes last in the severest way for days and weeks. To spasmodic conditions in the muscles of the pharynx and œsophagus we refer the well-known symptom of so-called *globus hystericus*—the patients feel as if a ball were moving up and down in the throat.

We sometimes see spasmodic conditions which come on in a clonic fashion, or in single twitchings in this or that muscular territory; they sometimes affect symmetrical groups of muscles, are not associated with disturbances of consciousness, cease during sleep, and are often easily cured by appropriate mental treatment. Such conditions have often been falsely called paramyoclonus, or myoclonia (*q. v.*). Among the forms of hysterical spasm we may also reckon the peculiar attacks, essentially of a psychical nature, when more complicated morbid excitations of volition occur, with or without the ordinary convulsive movements. In such attacks the patients begin to ejaculate the filthiest words of abuse (*coprolalia*), or to repeat every word they hear (*echolalia*), or to practice peculiar "impulsive" movements (tearing everything to bits, etc.). Conditions of this sort have been specially grouped under the name of "*maladie des tics*" (*vide supra,* page 603), but the greater part doubtless are hysteria. We see no reason to regard such conditions as a special disease under the name of "*maladie des tics*." We have repeatedly observed such conditions (especially in children), which were undoubtedly to be regarded as hysteria, and which could rapidly be made to disappear by appropriate psychical treatment. The severer forms of these conditions, of course, belong to the pronounced hysterical psychoses.

GENERAL COURSE OF THE DISEASE

Our description of the symptomatology of hysteria has been confined to the most important and frequent phenomena, and yet even this meager outline shows what an infinite variety of shapes the disease assumes. (1) In one class of cases there are no severe symptoms whatever. The patient merely displays the general mental condition characteristic of hysteria; she is easily excited, prone to make much of her ills, has all sorts of symptoms, such as pain, cephalic symptoms, palpitation, dyspepsia, and dyspnœa, and these are aggravated by mental excitement, while at other times they may so nearly vanish that the patient does not appear to be ill. (2) A second class of cases has more severe disturbance, coming on after some unfavorable psychical or other influence (*vide supra*). The patient may have displayed a general hysterical tendency previously, or may have seemed perfectly well. Here we may observe all the symptoms above enumerated and described. There may be paralysis, spasm, contracture, anæsthesia, or paræsthesia. The individual symptoms may persist obstinately for weeks and months, but again they may vanish suddenly or give place to other disturbances. Psychical influences are unmistakably potent, not merely in the incipient stage, but also in the further course of the disease. Any aggravation of the symptoms is often referable to emotional excitement. This is particularly true of the hysterical convulsions. In other cases, of course, the hysterical attacks come on apparently quite spontaneously, just like epileptic states. (3) The third class comprises the most severe forms of hysteria, with those nervous disturbances briefly outlined above. They are as complicated as they are puzzling, and form manifold combinations with all the other hysterical phenomena, including anæsthesia, contracture, and paralysis.

The entire duration of the disease varies greatly. The true root of all evil is the abnormal excitability of the nervous system, which always remains in unstable equilibrium, and often it is not possible to cure this. If not, the trouble lasts almost indefinitely. New manifestations of the disease succeed to periods of apparently perfect health. Usually the symptoms do not abate till quite late in life. There are, however, many instances of complete and permanent relief. This favorable termination is more especially to be hoped for where the patient comes into suitable and appropriate conditions of life, having some regular occupation which is not exposed to all sorts of unfavorable psychical influences. Many cases of hysterical disturbance, in previously healthy children or young adults, and due to some distinct cause, terminate comparatively soon, and never recur. It is never possible, however, to be sure that there will be no relapse, inasmuch as a single appearance of hysteria shows unmistakably that the nervous system is abnormally vulnerable to external impressions and the mental emotions excited by them.

Lastly, one more remark, which in my opinion is not unimportant. Since, as we have already seen, the foundation of hysteria is an abnormal congenital predisposition of the nervous system, it is a matter of little wonder if, besides the specific hysterical constitution, at times other constitutional abnormalities may be found in the same individual. Thus, hysterical symptoms are occasionally found in other diseases of the nervous system—for example, in multiple sclerosis, syringomyelia, tumors of the brain, all sorts of psychoses, etc.

Indeed, one can go even further and assume that in hysterical subjects, in addition to the abnormal constitution of the brain, other parts of the nervous system, or even entirely different organs, are functionally below par, an assumption which would naturally be of decisive significance for the pathogenetic conception of certain symptoms. It is impossible, in this place, to enter more in detail into these conditions. We wish merely to mention them in order not to merit the reproach of a too one-sided conception of hysteria. Nevertheless, I believe that an actual sharp limitation of the concept of hysteria is possible only on the basis of the psychogenetic nature of the clinical symptoms previously mentioned.

DIAGNOSIS

An experienced physician is seldom greatly puzzled by hysterical affections. Although the disease may at first simulate some grave organic disorder, a careful physical examination and continued observation will almost invariably disclose the true nature of the disease. In the first place, there are never any such symptoms as would absolutely prove the existence of some organic lesion. For example, we never find marked atrophy or loss of electrical reaction in connection with hysterical paralysis. We should also regard the whole psychical behavior of the patient, the influence exerted upon her by emotional disturbances, the disappearance of the symptoms when the attention is diverted, and the ætiology of the illness—for instance, the development of the symptoms after some mental excitement or emotion. The discovery of specific hysterical symptoms, the so-called hysterical stigmata, sensory anæsthesia, hysterogenous zones, etc., are especially important. That one must not go too far here has been emphasized above. Many symptoms of hysteria, especially hysterical abasia, hysterical aphonia, hysterical hyperæsthesiæ, certain forms of spasm, hemianæsthesia, etc., are in themselves so characteristic that from them alone a correct diagnosis can usually be made at the first glance. The very circumstance that hysterical affections do not keep to the laws of nervous anatomy and nervous physiology, and that they often cannot be brought into harmony with them, is of importance in their recognition; but, on the other hand, we must earnestly warn the physician against making the diagnosis of "hysteria" too readily. Pronounced hysterical symptoms are, of course, only rarely mistaken by an experienced physician, but very often all sorts of symptoms may be due to concealed affections, which cannot be positively determined (certain cases of gastric ulcer, gallstones, renal calculi, beginning cerebral diseases [especially multiple sclerosis.—K.], arteriosclerosis, etc.), and these symptoms may falsely be regarded as "hysterical," especially when we are actually dealing with nervous patients. Repeated careful objective examination and observation is therefore imperatively necessary. In doubtful cases the diagnosis can sometimes be rendered certain only by the result of treatment; but even in such cases the physician should not proceed too roughly or too rapidly, since he may only too easily cause harm.

Since an investigation of hysterical and epileptic attacks is of especial importance, we will give here a synopsis, somewhat schematic, of course, of the most important distinguishing features:

Epileptic Convulsions	Hysterical Convulsions
1. Sudden fall to the ground; often consequent external injuries.	1. Slow sinking to the ground; no external injuries.
2. Sudden pallor of the face. Often marked by cyanosis later.	2. No striking pallor of the face. No marked cyanosis.
3. Pupils dilated; do not react.	3. Pupils react.
4. Convulsions according to the nature of cortical epilepsy in the form of convulsive twitching. Tongue often bitten.	4. Convulsions consist of extensive movements, striking out with the arms or legs, twisting of the body, etc. Respiratory spasms. No biting of the tongue.
5. Often one initial cry, then total loss of consciousness.	5. Hysterical disturbances of consciousness, but no complete loss of consciousness; spasmodic and continuous cries, laughter, weeping, etc. Talking during the attack, emotional disturbances, delirium, etc.
6. Convulsions rarely last longer than a few minutes, followed by deep sleep.	6. Attack may last from half an hour to an hour or more. Patient often wakes suddenly.
7. Suggestive and hypnotic procedures, and also external irritation during the attack, without any influence.	7. The attacks may easily be excited artificially at any time by hypnotic or other suggestive procedures, or they may be inhibited in like manner (pressure on definite parts of the body or the like). During the attack the convulsions may be influenced or even wholly suppressed by sprinkling with water, a cold douche, etc.
8. Other hysterical symptoms are absent.	8. Other hysterical symptoms may coexist and often remain after the attack (hysterical hemianæsthesia, paralysis, contractures, etc.).

TREATMENT

What has been said about the ætiology of hysteria at once suggests a possible method of prophylaxis. A watchful eye will often detect, even in childhood, the signs of abnormal nervous excitability, and in such a case the parent will make it his duty to impose a suitable physical and mental regimen, that graver disturbances may be averted.

If hysteria be already developed, the first and most important treatment is mental. There could be no greater mistake than to deride the patient or treat her as a malingerer; for hysteria is a disease, and its symptoms are just as independent of any conscious volition on the part of the patient as those of any other disease. It is, however, also absolutely essential to carry out the moral training, which the physician must institute with all the proper strictness and energy, because in this way alone can any good be accomplished. Therefore an absolutely positive diagnosis is the first essential in the successful treatment of hysteria. If the physician is in doubt as to the nature of the trouble (*vide supra*), he cannot proceed with the necessary inflexible consistency, and the therapeutic results are seldom satisfactory; but if the diagnosis is assured, then there is no need of making the slightest concession to the patient. Sometimes this most important indication can be fulfilled only after the patient has been withdrawn from the overanxious and overassiduous parents or relatives, and like unfavorable influences. In such cases, treatment in some institution will often be vastly better than the best care at home, and our own experience leads us to recommend most urgently that the possible necessity of removal to an asylum should be constantly borne in mind with regard to aggravated cases. Often (e. g., in children) the mere dread of removal to such a place has a favorable mental influence.

Proper moral treatment achieves comparatively the best results where there is hysterical paralysis. When we are once certain that the paralysis is due to hysteria, the patient must be instructed how to regain by practice the lost power of the will over the paralyzed muscles. If the paralysis affect the lower extremities, as it usually does, the patient must be set on her feet, regardless of all her opposition and complaints, and kindly but most firmly required to try to walk. Of course, at first she must be well supported (see Fig. 223). This exercise must be methodically gone through with several times a day. Gradually the patient's gait becomes more and more secure. She regains confidence in her own ability, and, having once begun to improve, usually makes rapid progress toward complete recovery. Every experienced physician can recall numerous instances where hysterical paralysis which had lasted weeks and months was cured in a few days by this mode of treatment. Faradization of the muscles, cold sponging, and baths are excellent adjuvants; and the disagreeable element in these procedures of itself stimulates the patient to make every possible exertion to regain the use of her limbs.

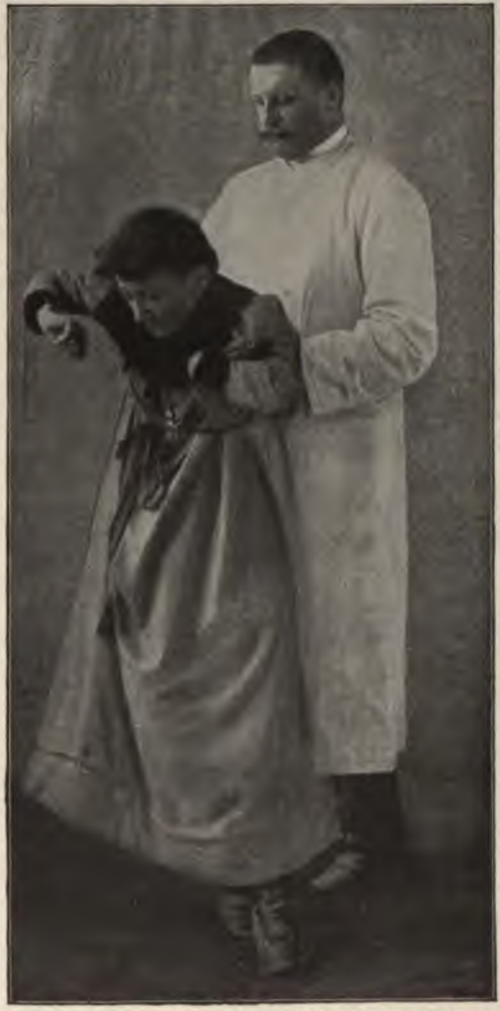

FIG. 223.—Hysterical abasia. Exercise in walking. (Erlangen Medical Clinic.)

When there is hysterical paralysis of the vocal cords, a similar training (energetic efforts to make the vocal cords tense and to produce a loud tone) will be found both practicable and efficient. Electricity is also of great value. It may be applied externally or within the larynx. Often the patient, terrified by the sudden pain it causes, recovers her voice at once. The same holds true of all hysterical disturbances of speech and of hysterical mutism.

The treatment of hysterical contractures consists, first, of an effort to relax the contracture by massage and energetic passive motion. Faradism will be found of assistance here also. In order to remove the contracture permanently, the patient must be induced to exercise the muscles regularly by making voluntary movements. We sometimes have the greatest difficulty in getting the patient to make the first attempt to move. In such cases we can attain our end only by great patience and perseverance and by the suggestive aid of all sorts of manipulations (galvanism through the head, etc.).

The treatment of hysterical spasmodic conditions often causes greater difficulties. The main thing is to bring the patient to control herself at the beginning of the attack, either by an energetic appeal, by diverting the attention, etc. In many cases a sharp sensory irritation, douching with cold water, or a bath with cold affusions, acts to restore the energy of the patient's will and the control over the muscles, and thus to check the spasms. The dread of the repetition of the bath does its part in restraining the patients from giving themselves up unresistingly to a repetition of the attack. The electric current (strong faradism during the attack) may also act favorably in the same way. Such measures, however, often gradually lose their efficacy; the patients become accustomed to the cold baths, and they remain without effect.

The milder varieties of hysterical convulsion, such as hysterical hiccough or cough, are often controlled by a stern reproof. It is precisely in these cases that the moral effect of transfer to some institution frequently causes the abrupt disappearance of symptoms which have lasted for months. All the forms of hysterical spasm occurring in children especially are easily removed by proper treatment (especially by isolation from the family!). The severe hysterical attacks, on the other hand, are often, of course, peculiarly obstinate, and they may resist for months, and even years, the most intelligent treatment. In all hysterical spasms limited to certain muscles methodical gymnastic exercises are often attended by the best results. We inhibit the abnormal motor innervations, and direct the will into other well-regulated paths—regular respiratory exercises in respiratory spasms, gymnastic exercises with the arms for twitching of the arms, etc.

Frequently the hysterical anæsthesias require no separate treatment. They belong to those symptoms which, as a rule, had better be entirely ignored. Then they often disappear of their own accord. If, however, in a particular case special treatment of the anæsthesia is desired, then electricity is most suitable. The anæsthetic areas of the skin are treated with the faradic brush, in order, so to speak, to recall to consciousness the anæsthetic portions of skin by means of the strong stimulation of the cutaneous nerves. It should be said, however, that these cases may prove obstinate or relapse.

The most difficult of all hysterical cases to treat are those where the symptoms are not strongly pronounced, but where there is a general hysterical condition, expressing itself in a multitude of nervous derangements, such as palpitation, dyspepsia, and general debility, or in purely subjective symptoms, or in emotional tendencies. Such patients are often advanced in years, so that little is to be hoped for from moral training; and their circumstances may be unfavorable without our being able to remedy the situation. Even here, however, the physician may greatly benefit them by means of psychical influences if he once gains their complete confidence. For patients of this class the treatment should consist principally of mental training and education. The patients should gradually learn to suppress their morbid ideas, and again accustom themselves to a normal way of living.

We should also consider all such remedies as invigorate the nervous system; electricity should be given, either in the form of general faradization, the faradic brush applied to the back and shoulders, or the galvanic current applied to the spinal column and the sympathetic nerve; and of still greater importance is a methodical cold-water treatment, either by sponging, bathing, or douches. Such patients are often vastly improved by sea bathing in summer, or by going to the mountains.

The numerous internal remedies for hysteria are also of more use in these general conditions than where there are marked nervous disturbances in special parts of the body. In the latter, internal remedies do good only indirectly and in a psychical fashion by so-called suggestion, especially if the patient have great confidence in the virtues of the medicine or in the physician who prescribes it. This is the explanation of the frequent cases of rapid recovery after taking homœopathic and "electro-homœopathic" remedies, and those still more marvelous cures effected by means of holy water and relics.

Among the "antihysterical" agents contained in the pharmacopœia, asafœtida, valerian, and castoreum are the most famous; but probably few would at the present day claim that they possess any specific virtues. Perhaps the preparations of valerian are the most useful in conditions of hysterical excitement, as evinced by convulsions or palpitation (pills of extract of valerian, gr. v to x [gm. 1.0 to 2.0] three times a day, or twenty drops of simple or ethereal tincture of valerian several times a day). Bromid of potassium, arsenic, and other medicines which ordinarily exert a favorable influence upon the nerves, seldom accomplish any permanent good in hysteria, although often prescribed. We must utter an urgent warning against narcotics, especially morphin, since they do little good, and there is great danger of developing the morphin habit in such patients.

If hysteria be complicated by some actual organic disease, the latter, of course, demands special treatment. Great benefit is hoped for by many from the cure of any uterine complaint which may be present. Cases are actually known where grave hysterical disturbance has vanished upon dilatation of a constricted cervical canal or rectification of a displacement; but there are numerous other instances on record where gynæcological treatment has proved entirely unavailing. There is hardly a doubt that, in the successful cases, the main benefit was due to the mental influence of the treatment. In general we would utter an urgent warning against needless gynæcological examinations and treatment in hysterical subjects. The patient's condition is often merely made worse by such procedures.

Accordingly, we may say with certainty that all methods of treatment of hysteria are efficient only when associated with the necessary psychical factor; that is, the patient's belief in the efficacy of the remedy, or, as we are accustomed to say at present, with *suggestion*—i. e., an effective influence on the ideation of the patient. Hence any prescription, no matter how senseless it seems, may have the greatest effect as soon as the patient "believes in it"—

that is, as soon as the psychical action of this belief is manifest. Hence it follows that in hysteria, as a rule, treatment either produces rapidly a brilliant success or it has no effect at all.

Two methods of treatment have still to be mentioned—metallotherapy and treatment by hypnosis. Very little is now heard of metallotherapy. We mention it merely as an historical example of what strange fruits the treatment of hysteria can produce. Hypnosis, however, still haunts many brains, whereby fact and fiction are often marvelously mixed.

In regard to metallotherapy a French physician, named Burq, discovered years ago that by laying plates of metal upon a cutaneous surface affected by hysterical anæsthesia a remarkable result is sometimes produced. Almost at once sensation is restored to the immediate region, and often to a much larger area. Most of the cases have been those of hysterical hemianæsthesia. Ostensibly, it is not every kind of metal which will prove effective, nor will the same kind affect all patients. It is said that iron is most frequently efficient; but sometimes copper, zinc, or gold is required. The process of determining the metal essential to each individual case Burq called "metalloscopy"; and he stated that this metal would also have the same effect if given internally! In 1876 a committee appointed by the Paris Société de Biologie tested these statements, at least with regard to the external application of metals—the idea of their internal administration having been pretty much abandoned—and confirmed them. Charcot also published many remarkable facts of a similar nature, which likewise soon received universal substantiation. The most remarkable of these phenomena is known as transfer. The return of sensation to the anæsthetic area, as a result of applying a metal plate, is accompanied by a simultaneous development of anæsthesia upon the opposite, previously normal, side, in an exactly corresponding place. Sometimes sensation oscillates from one side to the other and back again, so that now one half of the body and now the other is alternately sensitive or anæsthetic. If the metal be placed at the start upon the normal skin, that part becomes anæsthetic, while the corresponding part upon the opposite side of the body regains its former normal condition.

It has also been discovered that other hysterical symptoms exhibit analogous phenomena. Transfer can sometimes be observed in hysterical amblyopia, achromatopsia, deafness, loss of smell and taste, contractures, and paralysis. Such transfers may be induced by various means other than metal plates. These are classed as æsthesiogenous remedies, and include large magnets, feeble galvanic currents, and static electricity. Vibrating tuning forks and sinapisms have also produced similar results. Hence it seems to us to be an indubitable conclusion that the whole group of symptoms just described has no peculiar position. These symptoms are simply the results of suggestion, produced in this case by ideas, which are elicited through the peculiar method of examination of the patient. A specific metallotherapy is therefore entirely out of the question. We are dealing with pure psychical suggestion. The similarity of the test as performed by the physicians, who expect a definite result themselves, produces the similarity of the symptoms that ensue.

Of late the treatment of hysteria by hypnosis has acquired a far greater practical significance than metallotherapy; it is a mode of treatment which has been employed most extensively by the "school of Nancy" (Bernheim) and its adherents. If, during hypnosis, morbid states can be produced by suggestion, it naturally follows that morbid states can also be cured by suggestion. If, by many well-known successes, the hypnotizing physician has at the outset acquired the patient's confidence, as relics win the confidence of believers, the most beautiful results can, of course, be attained by hypnosis. There is no special peculiar principle in the hypnotic treatment. Any other efficient mode of treating hysteria rests on the same conditions and presumptions. Hypnosis has only the one great consequence, that it is artificially produced as a severe abnormal mental state in a patient who previously had not spontaneously fallen into this state. In that lies a consequence, which, of course, need not always be permanent, but which often enough has been most severe—the attempt to hypnotize a patient who is suffering from a slight hysterical affection has not infrequently been followed by the onset of a severe hysterical attack. The mischance will seldom happen to the physicians and magnetizers who practice hypnotism as a specialty, because their psychical influence on their patients is usually greater from the outset; but we should consider it a misfortune if hypnosis should come into too general use. That in this way apparently the most wonderful cures can often be obtained is perfectly well established, and not at all surprising; but the same cures can also be obtained in other ways, without running the risk of producing first that of which you would cure your patient, for to hypnotize means to make temporarily hysterical (see page 634), and there cannot exist the least doubt, therefore, that the use of hypnotism is a two-edged sword. The more frequently artificial hypnosis is employed, the more readily it can be produced, and thus it may happen that a hypnosis originally intended for therapeutic purposes ends in ordinary hysterical attacks. To this is added the circumstance that not infrequently the entire conscious state of the hysterical patient is by no means always favorably influenced by a hypnotic treatment with all the attendant claptrap. Furthermore, it is not hard to prophesy that, as the knowledge of the peculiar nature of hypnosis becomes general, it will lose its halo with the patient, and with that it will lose its healing power.

CHAPTER XII

NEURASTHENIA

(Nervous Debility. Nervousness)

Concept of the Disease and Ætiology.—The name of neurasthenia is given to a nervous symptom-complex which comes on in a great variety of ways. Its cause is not to be looked for in an organic disease, but in a merely functional disturbance of the nervous substance. The nature of this functional disturbance is ordinarily characterized by the two symptoms of "*abnormal irritability*" and "*abnormal exhaustibility*"; but the phenomena of neurasthenia and its allied morbid states are by no means wholly exhausted by the combined concept of "*irritable weakness.*" We may also add that the entire nervous system does not by any means take part in this abnormal functioning. On the contrary, the more carefully we study and analyze the symptoms of neurasthenia, the more the conviction is forced upon us here, just as in hysteria, that in neurasthenia we have to do essentially, although perhaps not exclusively (*vide infra*), merely with an abnormal mental condition of the sick person, and that the countless physical complaints and symptoms of the neurasthenic are ultimately to be referred in the main to a disturbed ideational life.

Nervousness and neurasthenia are brief practical terms applied to certain forms of an abnormal mental constitution. In by far the greatest number of all cases this abnormal mental constitution, or at least its basis, is congenital. Neurasthenia, of course, does not always show itself in childhood, although it does often enough. It does not always develop to the same degree, varying according to the action which various external influences exert in making it more or less prominent. We can only rarely, however, wholly dispense with the ultimate assumption of a definite congenital "nervous" predisposition. Of course we must admit that neurasthenic conditions of exhaustion and irritation may also occur under the influence of persistent mental overexertion or persistent and severe mental irritation in persons who were previously "perfectly well"; but in general we are usually inclined to attribute to these factors just mentioned merely the rôle of exciting causes, for every nervous system does not succumb to the same burden: one endures it without permanent harm, while another breaks down under it.

The congenital factor in the onset of neurasthenia is shown especially by a careful regard of hereditary conditions. Nervousness, neurasthenia, hysteria, degenerative insanity—these are closely associated morbid conditions, at the basis of which lies the inherited abnormal mental constitution, and these conditions are, therefore, unfortunately linked together in many families. Where the laity, from a natural dread of the worst forms, are disposed to believe in a fundamental distinction, the medical eye sees only quantitative differences in a long series of psychical conditions, which, without definite boundaries, join the normal psychical life on the one side and on the other pass into pronounced insanity.

It is our task in this place to describe merely the mild neurasthenic states, which far exceed in frequency all other nervous diseases. The idea that neurasthenia is a modern disease is entirely incorrect. It is as old as our definite knowledge of the diseases of mankind, and if the increasing frequency of neurasthenia is often ascribed to the restless haste, the unrest and the excitement of "modern" life, a glance at the history of past centuries with their horrors and their disorders of war will render such an hypothesis extremely doubtful. The name "neurasthenia" is the only thing that is new or modern. This, and the better insight into the nature of the disease, we owe to the American neurologist Beard (1880)[1] It is also true that all the exciting causes above mentioned are still active in their full force to-day, and that it cannot therefore amaze us if we observe neurasthenia with especial frequency in business men, whose bold speculations are attended by the excitements of hope and fear; in politicians, whose minds are constantly stirred by the eager strife of parties; or in those artists and scholars whose never quiet ambition forces them into restless competition. Neurasthenia develops often enough, however, in persons disposed to it from the first through a congenital defect, without any special exciting cause. We can readily understand that a disease, which plays its part chiefly in the ideational life of those affected by it, should be materially influenced by the degree of education of its victims, that is, by the richness and activity of their ideational life; but it is incorrect to call neurasthenia a disease chiefly of the "educated" or of the "higher classes." Neurasthenic conditions occur very frequently in the uneducated and in the lower classes, although they may manifest themselves in a somewhat different fashion.

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[1] [The disease had previously (1867) been described under the same name by Van Deusen, of Michigan, and the name itself is found in the first edition of Dunglison's Dictionary in 1833.—K.]

There is one point, however, yet to be considered. Even if we cling to the viewpoint of the psychical origin of most neurasthenic morbid conditions, it is still a question whether these "neurasthenic changes" cannot on their part be produced by entirely different causes. It is unquestionable that purely exogenous toxins can produce pronounced psychoses. Furthermore, many recent observations indicate that certain processes of "internal secretion" undoubtedly affect the processes of consciousness. We have but to think, for example, of the peculiar psychical symptoms of exophthalmic goiter, of the probable influence (little investigated as yet, it is true) of the internal secretion of the female sexual organs (ovaries, different processes during pregnancy and the puerperium) on the psychical functions, etc. It is consequently not impossible that certain cases of "neurasthenia" are due to disturbances of an entirely different character. Of course, hardly anything definite is known as yet on this subject. Further investigations in this direction, however, may be productive of important results.

The Symptoms of Neurasthenia.—1. THE MENTAL SYMPTOMS.—One of the most important changes in the ideational life of the neurasthenic is the frequent and ready appearance of ideas of an anxious character. So far as these ideas refer to the patient's own corporeity, they have been called from old times hypochondriacal. Sometimes they seem to arise of themselves, at other times they are produced by readily recognized causes (reading of histories of patients or of medical works, cases of sickness and death in the neighborhood, etc.). These ideas may constantly arise in consciousness, often in quite a fixed and definite form, and then dominate more or less all the rest of the ideational life. A large number of neurasthenics are dominated by the thought that they are threatened or already attacked by a serious organic disease. One constantly fears an impending attack of apoplexy, another heart disease, a third beginning consumption, a fourth believes he has noticed positive signs of disease of the spinal cord, cancer of the stomach, etc. The anxious ideas, however, may also take other forms; they sometimes refer to the patient's nearest relatives rather than to himself. Women are sometimes tormented by the constant dread that their husbands or children may fall sick. The ideas may also relate to external processes, with which the anxious ideas are easily and incessantly associated. The so-called agoraphobia (anxious ideas on entering a large open space), the dread of a crowd, of a railway journey, of society, the fear of sitting under the chandelier in a theater, the fear of fire, and many other symptoms of a similar character, are often observed in neurasthenics.

Not infrequently such ideas of an anxious nature come on suddenly with especial severity and lead to actual attacks of anxious distress with pronounced physical symptoms as a consequence. Such attacks may come on at night during sleep, drive the patient from his bed, and put him into a state of the greatest unrest and excitement.

We can readily understand that the ease with which these ideas of an anxious nature arise may keep the patient to some degree in a state of constant irritation. Hence such patients often complain: "I am always so restless—every trifle vexes me—everything, no matter how small, irritates me." Only when their thoughts are taken up by something else do they become quiet and behave in a way that seems perfectly normal.

These anxious ideas are by far the most frequent, but they are not the only form of morbid ideas which are observed in neurasthenia. In so far as such ideas constantly arise in consciousness independently of external events and constantly take possession of the patient, so to speak, we give them the general and fit name of *imperative ideas.* The following are some of the especially frequent forms of imperative ideas: Many patients are distressed by the constant recurrence of wholly superfluous questions: Why is this so—why is it not so? What would happen if this or that should ensue? etc. (*Grübelsucht*, metaphysical or reasoning insanity, *folie raisonnante*). Other patients distress themselves with constant doubts, with the constant feeling of uncertainty whether this or that has been done correctly, whether they have not written something wrong, have not forgotten something, etc. (insanity of doubt). These and other imperative ideas, which occur in a pronounced fashion only in the severe forms of neurasthenia, form the transition to the special forms of neurasthenic insanity.

Although the morbid ideational life in most cases gives the mental constitution of the neurasthenic the characteristics of excitement and restlessness, there may be also a pronounced inhibition of the rest of the ideational life, and consequently a depressed, melancholy disposition. The mental dispositions may sometimes vary in different forms. If there is in general an increased liveliness of the ideational life, the frequent and rapid transition from being "exalted to the clouds" to being "worried to death" may be especially striking.

The worst result of the disturbed ideational life, however, is the disturbance and inhibition of all other trains of ideas by the constant intrusion of the morbid ideas into consciousness. This is the chief reason why the neurasthenic is so often incapable of any persistent, methodical mental work, or at least why he can perform it only with the greatest effort. Sensations of bodily weakness (*vide infra*) are often added to the feeling of mental fatigue, among them being the inability to read or write continuously, sometimes associated with unpleasant subjective sensations of weakness or pressure in the eyes ("nervous asthenopia"). Of course in these disturbances of the power of mental work we must always make a sharp distinction between a congenital, primary, functional weakness, and an impediment to mental work due merely to secondary causes.

[The special morbid fears, like agoraphobia and the questionings and doubts, are rarely met with in neurasthenia, and are usually indicative of a pronounced neuropathic taint of a degenerative type. They may exist without neurasthenia. Anxiety, mental depression, and a tendency to worry are much commoner, and are of less serious import. Many neurasthenics, however, show hardly a trace of these mental disturbances, beyond a normal depression over their inability to perform the ordinary duties of life or their failure to regain their health. Their chief symptoms consist of inability to make any long-continued physical or mental effort and of physical weakness. As a result of overexertion various physical symptoms—headache, insomnia, backache, anorexia, indigestion, etc.—ensue, without any marked mental symptoms. Neurasthenia seems, therefore, to be rather a condition of pathological fatigue, upon which the various mental symptoms above described may develop secondarily. (See also the chapter on Psychasthenia.)—K.]

2. THE PHYSICAL SYMPTOMS.—Although the mental symptoms of neurasthenia are recognized only by the experienced physician as the special center of the disease, the neurasthenic himself, as a rule, claims to suffer from countless subjective symptoms, which may occur in almost every organ. By far the largest part of all these countless subjective sensations are, in our opinion, merely the result of the primary disturbance of the ideational life. We admit merely to the second place the possibility that there are independent functional nervous disturbances in other parts as well as the mental anomaly, and that these may lead to special physical symptoms. For, as we have already mentioned in the discussion of hysteria, it is conceded that the abnormal mental constitution may be associated with constitutional defects of other organs of the body. It is evident, however, that most of these subjective symptoms at least have a psychogenic origin, since they almost always conform absolutely to the character of the existing hypochondriacal ideas, and since they disappear entirely as soon as we succeed in driving these ideas out of consciousness.

In order to review the commonest neurasthenic symptoms we may very well classify them according to the organs to which the patient's fears generally refer them. The idea of a dreaded organic disease actually provokes the corresponding subjective sensations. Thus the "imaginary" diseases develop. If the patient's thoughts are occupied chiefly with the dreaded advent of a spinal disease, many symptoms apparently spinal ensue, especially pain in the back and unpleasant sensations along the spine, pain and paræsthesiæ in the legs, etc. The more the patient knows about actual diseases by experience or reading, the more manifold become his symptoms. Neurasthenic physicians are therefore the worst patients. As we speak of a "spinal neurasthenia," so we can also speak of a "cardiac neurasthenia," a "gastric neurasthenia," etc. The cardiac neurasthenics, who live in constant anxiety about heart disease and its consequences, complain of palpitation, pressure, pain and "fluttering" in the cardiac region, vertigo, and distress for breath. Patients with gastric neurasthenia ("nervous dyspepsia," *q. v.*) have a feeling of pain and pressure in the stomach after taking any food, and therefore are afraid to eat anything that is at all indigestible, suffer from unconscious voluntary eructations, etc. In cases of this sort it is indeed possible that we may be occasionally dealing at the same time with a true functional weakness of the organ in question. Nevertheless, by careful observation, the generally very predominantly psychical character of the affection is, as a rule, easily recognized.

The long series of physical neurasthenic symptoms is by no means exhausted, however, with such subjective symptoms provoked by definite anxious ideas (or by "*auto-suggestion*," as the modern technical term is). In almost all cases a large number of physical symptoms are apparent, which we regard as the necessary (that is, the physiological) result of the general mental irritation. As in every healthy man every great mental excitement is followed by a number of physical symptoms, it is plain that the abnormal state of excitement of the neurasthenic, coming on without sufficient external cause, may often have somatic results even in increased degree. Among these we may reckon first a large number of so-called "cerebral symptoms," such as headache, vertigo, and pressure and disturbance in the head, to which may of course be added many cerebral symptoms due to auto-suggestion. Among these belongs also the neurasthenic insomnia, one of the commonest and most important symptoms, and a direct result of the mental irritation itself. In many cases the insomnia forms the center of the neurasthenic symptom-complex. We may then say that the fear of insomnia is the chief reason for the actual insomnia. Many vasomotor symptoms are also a direct result of the general mental irritation—palpitation, a rapid pulse, redness or pallor of the face and hands, increased sweating or persistent coldness of the hands and feet, etc. We may also add tremor and the not infrequent increase of the tendon reflexes. Finally, the loss of appetite which many patients show, the habitual constipation, and in other cases the tendency to diarrhea, are certainly often most closely related to the psychical processes.

The liveliness of the motor innervations also depends in the main upon the general mental state. While many neurasthenics, impelled by constant restlessness, exhaust themselves in long and useless walks, there is in others a great physical weakness, which is due to inadequate cerebral volitional innervation of the muscles, so far as it is not dependent upon actual functional abnormality or impaired general nutrition. Such patients are soon tired on walking, they cannot work persistently, they sometimes feel so weak that they will scarcely leave their rooms, and they spend most of their time in lying on the bed or the sofa.

It is hard to decide whether neurasthenia exerts a direct influence upon the general bodily nutrition. We find neurasthenia in both full-blooded, corpulent persons and those who are frail and anæmic. Neurasthenia, however, very often exerts an indirect influence upon the patient's nutrition, since loss of appetite or fear of taking food gradually results in marked emaciation. On the other hand, we must always consider whether neurasthenia, as previously intimated, is not combined with other conditions of constitutional physical insufficiency. We may mention that large amounts of phosphates are often found in the urine, the reason for which is still uncertain. [In most cases of neurasthenia there is defective metabolism. The urine is scanty, the uric acid is increased relatively to the urea, and in sexual neurasthenia there may be an excess of indican. Many neurasthenics, however, drink very little water.—K.]

We must also devote a few words to a form of neurasthenia which is of great practical importance—sexual neurasthenia. It usually develops in persons who have practiced masturbation in their youth, and, by reading or their own reflection, have fallen into the most exaggerated anxiety as to the supposed evil consequences of this vice. Such pitiable patients usually fear that they are affected past help with some disease of the brain or spinal cord, and suffer accordingly from self-reproach and a host of corresponding subjective symptoms; but the very common "psychical impotence" is still more significant. This is a sexual impotence which is the result of anxious ideas which disturb the sexual act and make it impossible. Other sexual disturbances are usually added to these, which are at least in part a direct result of the physiological hyperexcitation which has occurred in youth (frequent pollutions, urethrorrhea, etc.). In sexual neurasthenia, therefore, the whole host of general neurasthenic symptoms above described are naturally grouped about this nucleus. Objectively, a certain flaccidity and smallness of the genital organs (penis and testicles) is not infrequently observed, which may be a result of a too early and too frequent use of these organs.

As has been indicated above, we are not justified in regarding all the physical symptoms occurring in neurasthenia as secondary psychogenous symptoms. Since neurasthenia is due to a general constitutional abnormal disposition of the nervous system, it is not strange if functional disturbances in other parts of the nervous system should occur along with the abnormal symptoms in the patient's ideational life. Thus, neurasthenia may be associated with true neuralgia, or with migraine or other forms of headache. It is therefore an important and often a difficult task for the physician to analyze the whole aspect of the disease in the individual case, to recognize the purely psychogenous symptoms as such, and to separate from them any coexisting physical nervous symptoms. This distinction is of great importance, especially in regard to the treatment to be advised. [It may also be important to determine how far the neurasthenic condition may be the result of some preexisting physical condition, which, by the attendant suffering, as of habitual headache, may have led to the general nervous exhaustion.—K.]

General Course and Prognosis of Neurasthenia.—The course of neurasthenia is governed in the main by the degree of general nervous predisposition that exists and the severity of the unfavorable influences that act upon the patient from without. If we are dealing with a patient who was previously in normal mental health and in whom a neurasthenic state of irritation or exhaustion has come on merely as an effect of unusual mental excitement or effort, such conditions of "acute neurasthenia" may wholly or mainly disappear under proper treatment and the removal of the injurious external influences. The condition is different in cases where the neurasthenic tendency has appeared quite early in the patient's whole mental nature. Here also we may often notice, of course, great variations in the prominence of the morbid symptoms; but an actual recovery is scarcely possible, since it would be equivalent to a complete change in the whole mental individuality—but in later life there may sometimes occur, even in such cases, a striking retrogression in the morbid symptoms. In the milder cases there is little outward evidence of derangement; the patient endeavors to hide his troubles, as his indefinite symptoms seldom gain much sympathy, and are apparently contradicted by his well-nourished and healthy appearance. In the severe cases, however, the patient's vigor is so much impaired that the disease acquires a grave aspect even for others than the patient, and fills them, as well as him, with infinite anxiety. The disease is never actually dangerous, and its existence only exceptionally prepares the way for more severe secondary nervous disease. Thus it happens that many neurasthenics, especially when external conditions are favorable, lead a comparatively endurable life and work hard and profitably in their calling.

Diagnosis.—Neurasthenia can usually be detected without difficulty, but the establishment of the diagnosis requires the exclusion of organic lesions of the nervous system and of other organs of the body. Every case, therefore, must be submitted to a thorough and careful examination. Grave cerebral diseases and psychoses, such as incipient tumors or general paralysis, melancholia, maniacal depressive insanity—furthermore, incipient multiple sclerosis, tabes, mild cases of exophthalmic goiter, etc.—have been frequently mistaken for neurasthenia.

It is, furthermore, by no means rare that patients with organic diseases of the internal organs, which are difficult to recognize, are incorrectly regarded as "neurasthenic." Thus, for example, many a case of cholelithiasis, chronic appendicitis, arteriosclerosis, gout, etc., is treated for "neurasthenia" for a long time, especially if the patient, as is often enough the case, is at the same time really "nervous." A great diagnostic danger, moreover, is to be found in the circumstance that all marked neurasthenic and hysterical patients may, of course, suffer from any form of organic disease, and that their condition is only too readily recognized. Consequently, as simple and easy, as the diagnosis of neurasthenia is for the experienced practitioner in most of the cases, we should nevertheless always examine the case anew without any preconceived opinion or prejudice. The fundamental diagnostic principle here, as in all other conditions, is to think of everything.

We cannot draw an absolutely sharp boundary, naturally, between neurasthenia, nervousness, and hysteria. In all these three conditions we are dealing, as has been said, with an abnormal nervous constitution, which is congenital or which has at least developed from outward circumstances on the basis of a congenital predisposition. This constitution is manifest in the first place in the purely psychical domain, and, in the second, in the relations between the mental and the corporeal life. If the abnormal state shows itself merely in slight mental irritability and sensitiveness, with a tendency to anxious ideas and unfounded fears, and thus to the development of all sorts of subjective abnormal sensations, referred, however, to the corporeity, we call it *nervousness*. If there is also associated a marked weakening of the capacity for mental effort and, in consequence of the defective or much inhibited mental energy, for physical effort as well—symptoms which, as a rule, are associated with increased anxiety and with consequent subjective sensations—we have pronounced *neurasthenia*. If, however, the relation between the mental processes and the corporeity has become so vacillating that there is a complete loosening of these relations (paralysis, anæsthesia), or that wholly abnormal states of mental irritation (spasms, attacks) have developed in consequence of the loss of all healthy mental inhibition, we have before us pronounced *hysteria*. The basis of all these three states is—figuratively expressed—the lack of all determined order and fixity in the course of all those processes which we include under the name of psychical processes in the broadest sense of the word.

Treatment.—As in hysteria, so also in neurasthenia, psychical or moral treatment is of prime importance; but here it must be a different kind from that necessary in hysterical cases. The neurasthenic requires sympathy. As soon as we recognize by means of a careful inquiry the hypochondriacal anxiety which is usually present, this is to be removed, as far as possible, by soothing persuasion on the basis of the thorough physical examination which has been made. The neurasthenic must be frequently examined and constantly encouraged and soothed. Herein, especially, is shown the physician's skill and personal influence. Every examination, which ends with the physician's assurance that he has found nothing to give occasion for any serious anxiety, has an extremely soothing and beneficial effect upon the patient. In so far as hypochondriasis is a prominent symptom, this moral influence may alone restore the patient to health. We should also pay attention to the patient's mental life otherwise. If the patient has been exposed to great strain and much excitement, we should, of course, procure a change. In such cases as these we prescribe mental rest, recreation, and abstention from work or any strenuous activity. Nothing, however, would be more improper than to try to make this prescription universal. On the contrary, it is absolutely necessary to prescribe resumption of regular and methodical work and occupation for many patients who, as a result of their mental disposition, do nothing but give way to their hypochondriacal broodings the whole day long. In this way alone can the thoughts of such patients be diverted, and the patients themselves be gradually brought back to take pleasure in their life and occupation. Of course we must beware of any overexertion.

Besides the psychical treatment, which must always take the first place, physical treatment is by no means to be entirely neglected. Although we are entirely of the opinion that the favorable results from physical methods of treatment in neurasthenia are to be referred in great, if not the greatest, part to their indirect psychical ("suggestive") influences, we, nevertheless, believe that attention to the general physical constitution is often a very important therapeutic aid in affecting the neurasthenic symptoms as well. All methods of treatment must therefore be used methodically and continued long enough for the patient to remain for some time under the permanent personal influence of the physician. In this way alone can the patient have that mental schooling and general improvement in the entire physical condition, which alone give promise of a permanent cure. What has been said explains why treatment in a sanitarium is so often desirable or even necessary for a neurasthenic.

Certain prescriptions as to diet are of importance in the physical treatment of neurasthenics. These are governed by the needs of the individual case. For a corpulent patient treatment calculated to diminish obesity will sometimes be followed by decided improvement in the general condition and in bodily vigor. In those frequent instances where the patient is pale and thin, and very likely oppressed by nervous dyspepsia, we should, on the other hand, make vigorous efforts to improve nutrition.[1] Definite instructions must be given in order that the patient may ingest a proper amount of food. Milk, butter, fresh meat, eggs, simple puddings, artificial foods, etc., are appropriate articles of diet. Often both the weight and strength improve rapidly. On the other hand, we must earnestly warn against a too one-sided valuation of these forced feeding cures. The mere increase of body weight, in itself, is not necessarily a therapeutic success; with existing hypernutrition, a restriction of the diet is even necessary. A too one-sided diet is especially to be avoided, above all an overabundant ingestion of meats and proteids. Fruits and vegetables are generally to be preferred. Any large amount of alcohol or of tobacco should be forbidden. Nothing is worse than to prescribe strong wine for a neurasthenic to "strengthen" him. We have countless times won a patient's gratitude by releasing him from such a prescription, which in his own experience he had found disagreeable (causing heat in the head, vertigo, etc.). Tea and coffee may be taken in moderation, if the patient is accustomed to their use. In regard to physical exercise, we must again be guided by the condition of the individual. We would most earnestly warn the physician against the error, frequently committed, of urging weakly and debilitated persons to take long walks. For such, bodily rest is much more desirable. Fresh air, which is certainly an advantage, need not for this reason be curtailed. On the contrary, we believe that rest cures in the open air, so-called sun baths, etc., are an excellent adjuvant to other methods of treatment. The sluggish and corpulent, on the other hand, often require persistent exercise. It is a good plan in many instances to employ the Swedish-movement cure, or similar gymnastic exercises. [In many cases the treatment must be directed primarily toward increasing the nutrition of the patient and building up the general strength in every possible way—forced feeding, cod-liver oil, tonics, etc. To this we must add rest and a change of occupation, or at least diversion. These are often more important than the psychical treatment, especially in the cases of pathological fatigue, where the morbid ideas are few or absent.—K.]

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[1] Playfair, Weir Mitchell, and certain other neurologists, have built up a special "method" of treating neurasthenia and allied conditions of nervous exhaustion; this consists in "overfeeding" the patient—that is, in introducing as large an amount of nourishment as possible into the system at the same time that complete bodily and mental rest is secured. Faradic electricity and massage are also employed daily. This mode of procedure is certainly excellent in many cases, but it must not be regarded as universally applicable. There are cases of neurasthenia for which it is not suitable.

Of special methods of treatment, electricity and hydropathy are at present most in use. Electricity is warmly praised by many patients. The galvanic current is generally employed, and is applied either over the sympathetic nerve or along the spinal cord. Its use demands great caution. The current should not be too strong, and there should be no abrupt changes in it. Galvanism applied to the head is seldom well borne. Another very valuable mode of treatment was first practiced by Beard and Rockwell, and consists of general faradization. The patient is almost completely stripped, and places both feet upon a large, flat electrode, while the various parts of the body are stroked with another large sponge electrode; in place of this second electrode the "electrical hand" of the physician may be employed. The physician takes the second electrode in his left hand and allows the current to pass through his own body. Various institutions have lately begun to employ electrical baths; these also often seem to produce good results. In addition to peripheral galvanization and faradization of the nerves and muscles, it is advantageous to employ the faradic wire brush, particularly on the back of the neck, along the spinal column, and over the shoulders and thighs. It is hard to say whether all these methods of electrical treatment have any specific action. The greatest part of the benefit obtained is undoubtedly of a suggestive nature; but there are few other methods by which such a suggestive effect can be so easily produced as by electricity, and therefore it is hard to dispense with it in the treatment of neurasthenia, in spite of what has just been said, since we often have to change our methods of treatment in so chronic a disease.

The hydrotherapeutic treatment may be in part carried out at the patient's home, but a severe case will be better off with methodical treatment in some well-conducted institution. Cold sponging, douches, hip baths, lukewarm baths (or sometimes swimming), are all employed. Douches must not be applied to the head. We are in the habit of prescribing half baths, with gradually reduced temperature, and douches to the upper part of the body. We cannot here give a detailed description of the different hydrotherapeutic methods. Detailed information may be obtained from text-books on hydrotherapy. The efficacy of hydrotherapy is due in part to its physical influence on the patient, which is undoubtedly favorable, but also, of course, in large part to suggestion, just as with all other forms of treatment. Instead of the plain baths, salt-water baths (neurogen baths), carbonic-acid baths, pine-needle baths, etc., are frequently to be recommended. Subsequently sea bathing is urgently to be recommended for many patients. We would recommend the seashore especially for emaciated and anæmic subjects, who are frequently greatly benefited by the improved appetite and rest thus obtained. If the patient be well nourished, on the other hand, a journey on foot through the mountains, if made cautiously, may be very valuable. Massage is also much used as a general means of strengthening in nervous conditions. We can judge of its value from what has been already said. It is prescribed most advantageously in those forms of nervous trouble which are associated with painful sensations in the spine, the extremities, etc. Massage displays an excellent direct effect when there is habitual constipation.

In neurasthenia, internal remedies should be given only as indicated by the symptoms or in the way of suggestion. The preparations of strychnin, quinin, arsenic, the valerian preparations, etc., are considered as the principal nerve stimulants and tonics. These drugs are generally prescribed in different combinations (tonic pills). If there is anæmia, iron, quinin, as well as arsenic (or subcutaneous injections of sodium cacodylate, etc.) are prescribed; if there is dyspepsia, some stomachic, such as dilute hydrochloric acid, pepsin, or some bitter. The constipation (see Vol. I, page 610) should be overcome mainly by diet, and laxatives should be given only in case of necessity. When there are vasomotor symptoms (a feeling of heat, congestion, palpitation), we often prescribe ergotin, 4 to 6 one-grain (gm. 0.05) pills a day. In all states of nervous irritation, valerian and bromid preparations are much used, a powder of potassium and sodium bromids, or bromin water. In nervous headaches, and also in other nervous states, antipyrin often has a good effect. Antifebrin and phenacetin, and many similar recent remedies, act in the same way.

The treatment of sexual neurasthenia is of great importance. As many patients of this class conceal their secret sorrows, it is advisable in all cases in which there is a suspicion of sexual neurasthenia to inquire in a tactful manner regarding this subject. It is a relief to most of the patients to ease their minds by discussing all their doubts and fears openly with the physician. The main thing is to enlighten the patient, and to remove his unnecessary worries. The general treatment of the neurasthenic symptoms is conducted entirely according to the rules previously laid down. If impotence exist, psychical treatment often produces the best results. Local hydrotherapy and electrical treatment are effective adjuvants. The drugs recommended as specifics (spermin, yohimbin, muiracithin), if they cure at all, act mainly by suggestion, but may nevertheless be tried. The question of marriage is a difficult one to decide. According to our experience a too conservative standpoint is not required. Frequently marriage is the best, possibly even the only way to effect a cure. If physical impotence continue after marriage, rational medical advice to both parties may still be attended by the best results.

The treatment of the insomnia which results from neurasthenia deserves a brief mention. It is usually a difficult problem. Since the insomnia is in most cases merely the result of the mental excitation, especially the onset of anxious ideas, the suggestive action of all the remedies that can be thought of plays, of course, the chief part, and the physician should take advantage of this circumstance. We must utter an urgent warning against the misuse of the stronger narcotics (chloral and morphin). The attempt should always first be made to secure sleep by a rational general treatment, or by some less injurious remedies. Often a warm bath at bedtime brings the patient sleep, and in other cases a brief cold foot bath or a wet cloth laid upon the head or back of the neck produces the same favorable result. Patients often report that general faradization at bedtime is an excellent soporific. Sometimes a moderate dose of alcohol may be tried as a true hypnotic. A glass of beer at bedtime often has a particularly good effect. If none of these means avail, our next resort should be the bromid of potassium. Very likely small doses of this have only a subjective effect, but there can be no doubt that a large dose, about ʒss. to j (gm. 2 to 3) in a glass of water, does have a direct tendency to produce sleep. We may also mention as the best of the other hypnotics veronal, gr. vijss. to xv (gm. 0.5 to 1); furthermore, sulphonal (poisonous in large doses!), gr. xx to xxx (gm. 1.5 to 2) in a large amount of water, in soup, [hot milk] or tea, two or three hours before bedtime. Trional is also to be recommended. These and other remedies, however, seldom give great satisfaction, and we must therefore rely mainly on general treatment.

CHAPTER XIII

THE SO-CALLED TRAUMATIC NEUROSES (ACCIDENT NEUROSES)

As an appendix to the last two chapters on hysteria and neurasthenia, we must now—more from practical than from scientific reasons—speak in particular of a series of morbid states which are at any rate most closely related to the above-mentioned affections, but which yet show certain peculiarities. The morbid states in question develop as the result of various sorts of physical injuries (trauma, accidents). They were first noticed after railway accidents (the so-called "railway spine" and "railway brain" of English and American neurologists), and it was formerly the belief that the coarse material concussion of the central nervous system was to be regarded as the cause of the subsequent nervous symptoms. Later, the name of "traumatic neurosis" was often used for these morbid states. By this we would imply that the nervous symptoms produced by the trauma do not depend upon coarse material injuries of the nervous system, but upon finer changes not yet made out anatomically. Soon, however, it was shown that, in at least the great majority of these cases, the material injury itself plays no other part than that of the exciting cause of a purely psychical change in the person affected by the injury. The material trauma is, as we express it, associated with a "psychical trauma." Among these we may reckon not merely the fright associated with the accident, but the whole series of new ideas occasioned by the accident and entering most vividly into the consciousness of the injured person—above all, anxiety about the further course of the injury, about the restoration of the capacity to work and earn a living, and also about obtaining any probable damages, etc. Sordid mental motives also are sometimes mixed with the others in consciousness, the desire for an accident pension, [for "damages,"] for a better way of living, etc. From what has been said in the two previous chapters we can easily understand that this primary change in the ideational life of the injured person may be followed by a considerable number of apparently physical disturbances.

The type of disease thus arising is peculiarly characteristic. Sometimes the original trauma (fall, blow, etc.) is so severe that directly after it the well-known symptoms of cerebral or spinal concussion (loss of consciousness, general motor paralysis, etc.) may ensue with more or less severity. In other cases, however, the intensity of the trauma or the special seat of its action (fall on the sacrum, back, or side, injury of a limb, etc.) is such that there can be no question of a material cerebral concussion. Not infrequently there may be some organic injuries at first (contusion, hemorrhage, fracture), but these of themselves are capable of perfect recovery.

The characteristic feature is that after the severe or slight initial material results of the injury have run their full course, there is not a complete cure, as might have been expected. The patient recovers, tries to get about again, and the improvement goes on up to a certain point; but, nevertheless, a number of symptoms remain which do not disappear, and which diminish or wholly destroy the victim's capacity for work. If we now examine such patients we find no signs of material injury either in the nervous system or in any other internal organ. What at once strikes us is a peculiar psychical change. The patients are dull, depressed, disinclined to any occupation, wholly without energy, and they have no more pleasure in intercourse with their families and friends. They brood constantly over their accident and its results. In very many cases the gloomy and listless disposition is mirrored in the patient's whole appearance, and especially in his facial expression. A practiced observer can often recognize the genuine "accident case" at the first glance (see Fig. 224). The sleep is disturbed and often rendered uneasy by dreams. Of subjective symptoms we must mention especially pains in the part which was hurt; these are commonest in the back, the sacral region, the sides of the chest, the hips, etc. Patients also complain of headache, vertigo, dullness, dim vision, tinnitus, spots before the eyes, loss of appetite, etc. Objective examination often shows a general motor weakness. Many patients can walk quite well alone, but they are soon fatigued; others walk only slowly and stiffly with support, complaining of pain in the back on walking, and therefore holding the hand on the back, etc. The nutrition of the muscles is usually good. The electrical excitability of nerves and muscles is completely normal. The reflexes are sometimes lively and sometimes weak, but never so much changed as we find them in actual organic diseases. The pharyngeal reflex, as in almost all forms of hysteria, is frequently markedly diminished. Examination of the sensibility may reveal very marked changes, which do not occur in all cases. This examination must always extend to all the sensory organs. The skin over almost the entire body is often very insensitive to painful irritations, pin pricks, the electric current, etc. There is analgesia. In some places we find complete anæsthesia, which may affect an entire extremity or only circumscribed parts of the extremities and the trunk. The boundary between the anæsthetic and the normally sensitive parts is indistinct or quite sharply defined, and the arrangement is often very peculiar, in a way that can never occur in organic nervous diseases. Examination of the eyes sometimes shows loss of visual acuteness, limitation of the visual field, and imperfect color perception. Very often the hearing is poor on one or both sides. The smell is often much blunted, and the taste completely lost, so that even quinin, vinegar, and substances of equally strong taste no longer excite any sensation of taste. All these sensory anæsthesias (unless perhaps simulated) must be regarded as hysterical anæsthesias. We are justified in assuming, therefore, that frequently they are merely developed by the examination—i. e., the medical examination disturbs the consciousness of the patient in such a manner that a sensory anæsthesia is developed. The physician should always think of this in the examination of accident cases. [Although this possibility should be borne in mind, the danger of suggesting anæsthesia, etc., if the examining physician be skilled, has been greatly exaggerated.—K.]

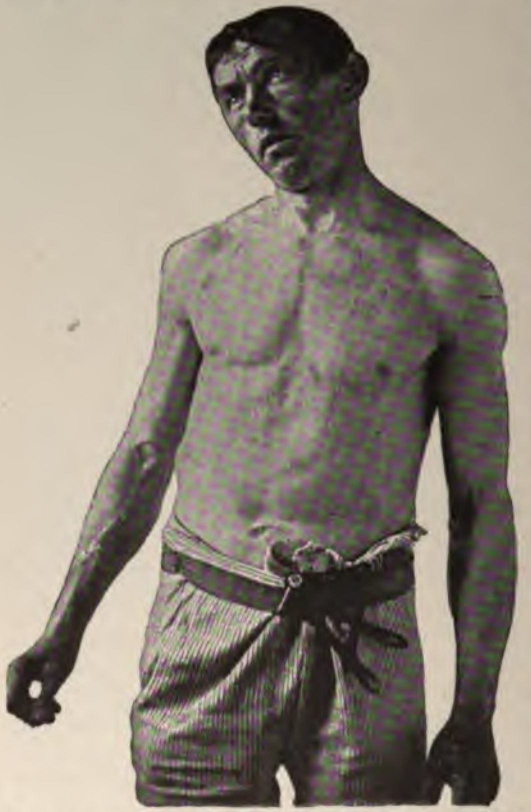

FIG. 224.—Accident case, with hysterical paresis and tremor of the right arm (personal observation).

Besides the symptoms of impairment of sensation and motion just described, we often find also symptoms of sensory and motor irritation. Pain in the head, spots before the eyes, and tinnitus have been mentioned above. Hyperæsthesia of the skin is more characteristic—tenderness of the vertebral column, and particularly great tenderness of such parts of the body as were most affected by the original injury. Among the symptoms of motor irritation we may mention muscular contraction and muscular rigidity, again most common in the limbs most affected by the injury. A marked tremor is also very common.

Almost always the subjective symptoms are referred definitely to the spot which was first injured. If the injury has affected the head, the chief complaint is there; if one side of the trunk or one limb has been affected, most of the symptoms are manifested there, but of course the symptoms often extend beyond the part originally injured. If, for example, one shoulder receives a blow, the whole side, including the leg, etc., is often weak and painful. General nervous symptoms, especially the subjective feelings of vertigo and pressure in the head, are found in the majority of cases, even after peripheral injuries.

If we inquire into the cause and the special nature of this peculiar affection, characterized chiefly by psychical disturbances and in the second place by subjective sensations of pain, sensory anæsthesia, and motor weakness, the purely material effects of the injury are in most cases not to be considered, as we have said. Although we at present believe that there are morbid states which we must actually regard as traumatic neuroses in the stricter sense of the word, and as permanent functional disturbances resulting from an actual material cerebral concussion, the assumption does not hold for the vast majority of all "traumatic neuroses," for the neurosis is certainly produced by the *ensemble* of psychical effects, associated with the trauma (accident), which have been described in detail above. The perplexity and frequent contentions about sick funds, insurance societies, and claims for damages, do their part in keeping the patient disturbed. In laborers who have accident insurance, and in whom the "traumatic neuroses" are far most frequently observed, we often find an intense desire to obtain a very high pension, and, moreover, a peculiar obstinacy in their claims and their opinions in regard to a presumed right of damages after every accident. We may therefore assert that the traumatic neuroses have become decidedly more common as a result of the accident legislation.

[In America claims for "damages" against corporations are much more important factors in the ætiology of the traumatic neuroses than any claim for insurance. The various psychical influences associated with the litigation consequent upon such claims have given rise to the term "litigation neuroses." Although it is undoubtedly true that litigation aggravates, and sometimes perhaps originates, nervous conditions, it is equally true that we often see precisely similar conditions developing after an accident in which there was no possibility of making any claim for damages or insurance, and where the other psychical factors above referred to had only a slight influence. Furthermore, on considering a large number of cases, there seems to be some relation between the severity of the physical injury and the severity of the subsequent nervous conditions, even in the cases of genuine hysteria, although hysteria may at times develop after a very slight physical injury. The pain and other physical effects of the injury may, of course, if long continued, so far affect the general condition as greatly to aggravate the nervous sequelæ, but the influence of the actual material concussion is often underestimated, and the "accident case" often receives less consideration than he really deserves.—K.]

Various factors therefore act in producing a condition which has the closest points of contact with general nervousness, neurasthenia, hypochondria, and hysteria. It would therefore be proper, now that our views as to the nature of the traumatic neuroses—a term, by the way, which we have always used merely in a symptomatic significance, and therefore justifiably—have become clear, to use this term merely as a symptomatic appellative and replace it by the terms "traumatic neurasthenia," "traumatic hysteria," etc. On the other hand, we must not forget that the neurasthenic and hysterical affections following accidents often have a certain peculiar stamp, a common type which we do not often find in ordinary hysteria and neurasthenia. The chief characteristics are the patient's peculiar psychical state, manifestly conditioned by the special external circumstances, his lack of courage and energy, his depression, the tendency to complain and be querulous, and also the great obstinacy with which these conditions persist and often resist any attempts at treatment. We know many cases where such patients, in spite of the fulfillment of every desire, have lived for years in a state of permanent neurasthenia, incapable of anything, and finally have fallen into a state of pronounced mental dullness. We may therefore still use the term "traumatic neurosis," or, better, "accident neurosis" or "accident psychosis," for such cases from practical reasons, if we only associate the correct meaning with it. In all those cases, however, where there are pronounced objective symptoms of hysteria (anæsthesia, paralysis, contractures at the seat of the injury), we should speak merely of hysteria with the appropriate adjective, "traumatic" hysteria.

In the great majority of cases it is not at all difficult to recognize positively the "traumatic neuroses." The existence of gross material injuries can, as a rule, be easily excluded. It may be harder to decide whether there is real disease or simulation. In such cases we cannot decide by single criteria—as has been supposed—but ultimately only by careful observation of the constitutional state (if possible, in a hospital) and by consideration of the general conditions (the patient's behavior, previous life, external circumstances, etc.). From our own experience we must in general state that conscious and intentional complete simulation is not very common, although, of course, it occurs often enough. Exaggeration and apparent simulation are, of course, very common, since the patients report symptoms which careful investigation shows to be absent, or at least not to be constant. Such a condition, however, is no proof of conscious simulation, since we see it every day in all the psychoneuroses (hysteria, neurasthenia). An hysterical woman who claims that she cannot leave her bed, and then walks very well when led by the physician; another who says she cannot bear the slightest touch on her back, and then hardly feels the strongest pressure when her attention is diverted; a third who claims to have pain in the stomach after a swallow of tea, and soon after eats a beefsteak with relish—would not be called a "simulator" by any physician. A similar thing occurs, of course, in the accident neuroses, because they are precisely analogous conditions. It is of course very hard to decide the question, which is often of such practical importance, how far such neurasthenic-hypochondriacal patients have a claim for damages and pecuniary assistance. In such cases we are decidedly of the opinion that the examining physician should not be too generous, since it is necessary from a therapeutic point of view to cure the patient of his delusion that he is suffering from a serious disease. We should therefore always try in the first place to bring about an amelioration or a recovery from the condition, and of course the chief stress is to be laid upon the proper psychical influence to be exerted on the patient; but we must also consider all the other methods of treatment (baths, sponging, electricity, gymnastics) with which we have become acquainted in the two preceding chapters. In this way we sometimes succeed undoubtedly in bringing about improvement and even recovery, but the outlook for the permanent success of the treatment is not in our experience very good. The accident neuroses apparently have an especially unfavorable prognosis, although, as we know, the same is unfortunately often enough the case in non-traumatic states of severe neurasthenia and hypochondria. In the accident cases, however, we often notice from the outset that they do not wish to be treated or cured. The incurability of their condition has become a matter of conviction to them, and their only interest is to utilize the accident to the utmost—i. e., to obtain as large an accident annuity as possible. In most cases, at least, we should therefore advise against any continuous medical treatment unless we have the impression that the patient will submit willingly and trustingly to the physician's directions. It seems to us of the greatest practical importance to "strangle" the traumatic neuroses "in the embryo," if possible, on their first development. Not infrequently we have seen accident cases who began to complain constantly of all possible nervous troubles after some slight external injury was evidently wholly cured. In such cases we should cut short such complaints as completely as possible from the outset and make the patient understand that he is now actually and completely well. The granting of a high accident annuity at first is therefore quite incorrect in such cases and makes recovery much harder, while the immediate and complete refusal exerts the best influence. The patients are forced to resume their work and to forget their imagined symptoms. If we, nevertheless, believe in awarding damages to the patient, it is preferable to grant a fixed sum than a continued pension; for, through the repeated medical examinations required in the latter case, combined with the ever-existing wish on the part of the patient not to lose the pension which he has secured, the cure of these accident neuroses is rendered very difficult, or almost impossible. If a traumatic neurasthenia has continued for years, it is usually very hard to convince the patient that his complaints have no objective foundation.

[In many cases the patient is actually injured by resuming work too early. It is sometimes of decided advantage, especially in the early stages of the trouble, to prescribe a thorough "rest cure," with isolation, massage, forced feeding, etc., as prescribed by Weir Mitchell. The worries of litigation should be eliminated, if possible, by an early settlement of the case. It must be borne in mind that the various therapeutic procedures, which are often advisable in neurasthenic and hysterical conditions, such as massage, electricity, gymnastics, good nursing, travel, etc., are extremely expensive and often impracticable for those in poor circumstances. This is of importance when the physician is called upon to determine the amount of the damages or the accident annuity.—K.]

VI. MENTAL DISEASES[1]

CHAPTER I

CONFUSIONAL INSANITY

(*Amentia. Toxic and Infectious Psychoses. Alcoholic Insanity. Acute Delirium. Acute Hallucinatory Paranoia. Sensory Phrenosis.*)

Ætiology.—The most important cause of this affection is the action of some poisonous substance upon the brain. This poison may be taken into the system from without, as in the cases due to alcohol, lead, opium, and other poisons, or it may be formed within the body—auto-intoxication. In the latter case it may be formed in the course of some infectious disease, such as influenza or typhoid fever, or be the result of somewhat obscure changes in metabolism, as in disturbed activity of the thyroid gland, cancer, diabetes, or gout. Confusional insanity may also develop in the course of diseases of probably toxic origin, such as pellagra, or even, in rare cases, as the result of auto-intoxication arising from chronic constipation. Before aseptic midwifery was practiced a considerable number of cases arose from puerperal infection ("puerperal insanity"). Profound exhaustion and malnutrition may give rise to the disease, but in such cases we must again consider the possibility of auto-intoxication (toxins of fatigue?). In some cases, of course, it is difficult, if not impossible, to determine absolutely any toxic cause.

Since the various toxic and infectious influences referred to above may act at some time or other upon the majority of individuals, it is evident that some other factor must also be called upon to explain the special incidence of the disease. That factor is individual predisposition. The existence of a neuropathic predisposition in the family—that is, an unusual frequency of occurrence of the various forms of nervous and mental disorder, of alcoholism and drug addiction, of marked eccentricities of character, of criminal tendencies, etc.—is an important element in rendering the individual more susceptible to the action of these toxic and infectious influences. If, too, certain injurious influences—alcohol, infection, malnutrition, etc.—have affected the parents during the period of conception and pregnancy, the offspring is likely to be enfeebled and more susceptible to various ills in later life, especially to affections of the nervous system. Other factors are of less importance, but they sometimes seem to have an influence in the development of the disease. Among them are injuries, especially injuries to the head, mental or physical shock, worry, and emotional strain. In many of these cases we may query as to the development of some auto-toxin as a result of the shock or strain.

Symptoms and Course.—The essential feature of the disease, as the name implies, is mental confusion, which is often attended with hallucinations, especially of sight and hearing, and with a greater or less amount of delirium. In many cases there is no disturbance of any form of sensation; the patient can see, hear, or feel in a perfectly normal fashion, and even if there be some sensory disturbance, as in alcoholic neuritis, it is not sufficient to explain the confusion. The difficulty is not with the simple perception of sensations, but with the understanding and recognition. There is a loss of association of ideas and of normal coördination of cortical images, which has been sometimes spoken of as intrapsychical ataxia. Everything seems unreal; when the patient succeeds in recognizing persons or places they seem different, and often he fails to recognize them altogether. Although he is in his own room at home, he will insist that he is far away in a strange place, and must get back, or he may believe that strange attendants are old friends. His ideas as to times and seasons are often erroneous; he cannot tell the day, the month, or the time of year. He sees visions and hears voices, which may be agreeable or alarming. With this there is a marked failure of ability to pay attention to any subject, and to keep his mind fixed upon any matter. He may be able to pay attention for a moment, but he soon begins to wander. He is unable to talk connectedly, or to answer questions consecutively; his mind wanders and his talk becomes confused, disconnected, and, finally, a string of words associated merely by similarity of sound or rhyme. Delusions of various sorts may develop, sometimes as a result of the hallucinations in an attempt to explain them. These delusions often refer to some attempted persecution, which is manifested by threatening voices or sights. Owing to the failure of attention the patient's ability to understand is materially lessened, and the memory is somewhat impaired.

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[1] [P. C. K.]

When the disease is fully developed it is apt to manifest itself in one of two forms. In one form the hallucinations are marked, various delusions develop, which, however, vary from time to time and seldom become permanently systematized, and the patient reacts to them in accordance with their character. If they be alarming, as is apt to be the case, he seeks to escape or resist, he becomes violent and destructive, makes loud outcries, attacks his nurses, and is actively delirious, raving, struggling, noisy, and maniacal. In the other form the hallucinations and delusions are less pronounced, the patient is confused and wandering, and more stupid. He can be roused, and will answer questions, but his attention is hard to fix; he talks little, often mutters to himself, and may grow more and more stupid, so that he becomes unable to feed himself or notice the calls of nature. In the severer cases both forms may terminate in a grave typhoidal state of profound exhaustion, with muttering delirium, subsultus tendinum, and a stuporous or even comatose condition, from which the patient can be roused only with increasing difficulty.

In addition to these mental symptoms there are certain somatic symptoms which are apt to be present. In the early stages the patient may complain of headache, easily induced fatigue, sleeplessness, loss of appetite, inertia, and inability to use his mind. The sleep and appetite are usually disturbed throughout the active period of the disease, and when the patient does sleep, he is often the victim of unpleasant or frightful dreams. The nutrition is more or less profoundly affected. The weight decreases, the temperature is apt to be elevated at first, but later may fall below normal. The urine often contains a trace of albumen, the patient grows anæmic, the reflexes are at times diminished, and there may be some slight diminution in sensibility. In the cases due to pellagra other symptoms of pellagra are, of course, present. (See page 708.)

The disease may develop somewhat gradually with a prodromal period in which some of the milder symptoms above-mentioned predominate. The patient is nervous, restless, and uneasy, and suffers from headache, loss of appetite, sleeplessness, and bad dreams. He is anxious or apathetic, and complains of inability to concentrate his thoughts or to persist in any mental or physical work. This same condition is often seen in febrile states, but then it may be of short duration. Later the mental confusion and hallucinations become apparent, as they do in ordinary febrile delirium. In the febrile infectious psychosis, however, the mental state may not develop until after the fever begins to decline, or it may form a continuation of the febrile delirium. In some cases the active delirium develops suddenly, soon becomes severe, and may prove fatal in a few days (*delirium grave*). In such cases there is a grave typhoidal state, high temperature, and an active violent delirium. In other cases the period of mental confusion and hallucinations may continue for several months, or even a year, gradually diminishing until the patient recovers. He may never fully regain his former mental power, and often shows a defective memory and appreciation of the period of his illness. In other cases, still, the disease diminishes, but the patient remains with permanent mental impairment, either in the form of a delusional, paranoid state or of dementia. During the course of the disease lucid intervals are said occasionally to occur, but these are rare. There may also be alterations in the mental state, and periods of apparent improvement followed by an exacerbation of the condition. In cases of long-standing pellagrous insanity there may develop a condition of pseudo-paresis, with profound dementia, euphoria, and motor symptoms (disturbances of speech, exaggerated reflexes, and immobile pupils).

The type of the disease, as has been said, may differ, and all sorts of transitional forms may occur. The hallucinations and delusions may be so marked as to conceal the confusion almost entirely (acute hallucinatory paranoia), or the confusion may be the chief feature, the hallucinations being scarcely noticeable. Again, the disease may take the form of a very intense febrile delirium, as has just been described. The various syndromes which may develop as a result of alcohol indicate that these different clinical forms may fairly be grouped under one heading. The alcoholic affections, however, present certain individual peculiarities, and merit a special description.

1. ALCOHOLIC INSANITY. (*Delirium tremens.*)—This does not develop as the result of a single indulgence in an excessive amount of alcohol, but from continued abuse. The patient, after a period of sleeplessness, loss of appetite, gastric disturbances (morning vomiting), and headache, begins to develop marked tremor and persistent insomnia. He is restless and anxious. Hallucinations soon develop, especially in the visual sphere; next, hallucinations of touch, and, rather rarely, hallucinations of hearing. The visual hallucinations at first seem merely black, moving spots, but later they assume the form of insects or small animals. The patient becomes more and more fearful and restless. He talks rapidly, tries to evade the animals, and is often resistant and violent. He can pay attention to a subject proposed for a few moments and seem rational for a time, but soon is distracted, and becomes more and more delirious. Consciousness is not disturbed, but his ideas of time and place are confused and the external world seems unreal. His memory for past events, however, may be fairly well retained. The fever increases, the patient eats little, and sleeps not at all; the hallucinations grow more vivid

and alarming, and the delirium more violent. The pulse is rapid, the temperature elevated, and there is usually albuminuria. Pneumonia is a frequent complication. The condition may increase to a stage of low, muttering delirium and increasing coma, and death ensues from cardiac weakness. If the patient can be made to sleep, recovery is often rapid, but the active delirium may pass into a state of confusion, the so-called Korsakow's psychosis.

2. ALCOHOLIC CONFUSION (*Chronic Alcoholic Delirium. Korsakow's Psychosis*).—This has already been referred to in the chapter on alcoholic neuritis, but it may develop independently of neuritis, either primarily or secondarily to an attack of delirium tremens. The condition often begins with a delirious stage of greater or less severity, but the delirium, while less pronounced, may be of longer duration than the ordinary delirium tremens. Then it is noted that the patient is dull, that the attention is defective, that he has a defective power of receiving new impressions, and that he has lost memory for recent events. He is also decidedly disoriented—that is, he is unable to recognize his surroundings, or to tell the day or the month. Often he may appear to understand what is said and to talk fairly intelligently, but clever inquiry will show a tendency to confabulation; he will tell a long story of where he has been that morning, whom he saw, and perhaps how many drinks he has taken. He may have some hallucinations, and is often restless, noisy and uneasy, especially at night. Often he is somewhat stupid, and passes urine and fæces half unconsciously. His power of attention shows the defect above described; he can pay attention for a few minutes, but soon grows inattentive. If the attention can be retained, however, the processes of thought may still be carried on fairly well, and there may be no actual intellectual failure. The process of recovery often shows a somewhat curious return of memory; the patient remembers that he has not been out to-day, but he tells the same story of his travels yesterday, then the day before yesterday, then last week, until finally he regains a correct estimate of time, although the period of his illness often remains hazy or a complete blank.

3. PSEUDO-GENERAL PARALYSIS OF ALCOHOLIC SUBJECTS.—Just as we speak of pseudo-tabes, so we may use this term for a terminal stage, not infrequently seen in those who indulge excessively for a long period, which may follow either delirium tremens or the confusional state. The mental confusion and failure of memory, attention, etc., increase until the patient becomes quite demented. Hallucinations and delusions may persist, but they are vague, and not systematized. The patient becomes more and more dull and stupid, is unable to care for himself, soils himself unwittingly, wanders about undressed, and is unable to feed himself. To these are added various motor symptoms, due partly to neuritis, partly perhaps to arteriosclerotic processes in the brain and cord. The gait is weak and unsteady. All the movements show tremor and incoördination. The speech is indistinct, and there may be stumbling over syllables, as in true general paralysis. The handwriting at times may closely resemble that of a general paralytic. The pupils, however, react normally, the cerebro-spinal fluid does not show leucocytosis, and the Wassermann reaction is negative if the patient be not also syphilitic. The condition increases until the patient becomes helpless, bedridden, and finally comatose. These are the cases that show the most marked œdema of the pia—" wet brain."

4. ALCOHOLIC HALLUCINOSIS (*Alcoholic Paranoia*).—This form of al-

coholic disturbance usually develops primarily, without any previous delirium. The patient may present the ordinary symptoms of chronic alcoholism before the onset of the disease—insomnia, tremor, gastric disturbances, etc.—and he often seems more sensitive to noises, and may complain of some tinnitus or noises in the ears. The first striking feature of the disease is the development of hallucinations, which are much more frequently auditory, unlike the hallucinations of delirium tremens, although visual hallucinations also occur. Soon after this delusions develop, which are usually referred to the hallucinations and attempt to explain them. These rapidly become systematized and quite elaborate. The patient hears people talking about him, calling after him and abusing him. They may threaten him or accuse him, and this is because he is the victim of some persecution. In other cases the delusions may be of a religious character—the patient believes he has committed unpardonable sins, sees God threatening him, or devils coming to take him to hell. His thoughts are read, and he is influenced by concealed wires or wireless telegraphy. One of the common forms of delusion is that of jealousy and marital infidelity, excited possibly by the diminished virility that follows alcoholic excess. The husband suspects his wife of infidelity, and tells detailed and elaborate stories of acts of open licentiousness. As a result of these delusions the patient is anxious, depressed, fearful, and at times violent, or sometimes mute in response to a delusion. With all this there is little or no confusion, the attention and memory are good, except as affected by hallucinations, the intellectual activity is nearly normal, and the patient is clear and well oriented—that is, he has correct ideas of time and place. Under favorable circumstances this condition may gradually disappear in the course of a few weeks, but sometimes the patient may remain in a chronic paranoid state, or, if alcoholic indulgence continue, may develop the terminal pseudo-paralytic state ("wet brain") just described. As already indicated, these alcoholic states may pass into one another, and various transitional forms may occur.

Pathology.—The pathology is still obscure. In many cases, due to infection or poisons such as alcohol, there may be various changes elsewhere in the body due to the infectious disease or to chronic alcoholism. Apart from these the brain itself is often hyperæmic, sometimes intensely so, and occasionally shows punctate hemorrhages. In alcoholic cases these hemorrhages may be of considerable size, but they are to be regarded as merely complications. The chief changes referable to the disease itself are minute alterations (chromatolysis) in the cell bodies in the cerebral cortex, especially in the large pyramidal cells, and degeneration in the axis-cylinder processes of these cells. In cases of chronic alcoholism particularly, and in all the chronic cases to a lesser degree, we find thickening of the pia mater, milky opacities along the pial vessels, more or less œdema of the pia, some atrophy of the convolutions, and more or less arteriosclerosis of the cerebral blood vessels. In alcoholic cases the œdema of the pia may be so great as to justify the term "wet brain." Bianchi and Piccinino claimed to have found a specific bacillus in the very severe cases of acute delirium or *delirium grave* (Bell's disease), which they believed to be the cause of the disease, but their hypothesis has not been confirmed by other observers.

Diagnosis.—The diagnosis must be based upon the ætiology, the history of some infection or intoxication preceding the outbreak of the disease, and the existence of mental confusion with hallucinations, with the loss of power to associate ideas, and to coördinate cortical images. If the confusion be marked, and there be much excitement, the disease may be mistaken for mania. In mania, however, the confusion is more apparent than real, and is due to the failure of the patient to express his ideas fast enough. It is manifested only under conditions of excitement, while, in confusional insanity, the confusion is evident when the patient is quiet. Cases with marked hallucinations and delusions may easily be confused with true paranoia. In paranoia, however, we usually have the history of congenital mental peculiarities, and a gradual development of the delusional state instead of an acute onset. It is only rarely that cases may be mistaken for general paralysis. The somatic symptoms—pupils, reflexes, speech, and handwriting—and the examination of the cerebro-spinal fluid will usually give definite information. A very similar clinical picture to Korsakow's psychosis may be presented by that form of senile insanity known as presbyophrenia. (See page 695.) The age and the lack of any history of alcoholic excess are the chief aids in distinguishing this from alcoholic confusion. The distinction from dementia præcox is difficult, and will be discussed in the next chapter.

Prognosis.—The prognosis varies with the different forms of the disease. Cases of active delirium with high fever afford the most serious prognosis, so far as life is concerned. The mortality usually exceeds fifty per cent. The prognosis is governed by the height of the fever and the severity of the general condition. The outlook in the ordinary case of confusional insanity is fairly good; more than one half the cases recover, although the trouble sometimes recurs. The prognosis in delirium tremens is rather grave; the mortality varies from ten to thirty per cent, and, if pneumonia develop, it is considerably higher. The patient usually recovers from the first attack of alcoholic hallucinosis in a few weeks or months. The outlook in alcoholic confusion is more serious. A number of writers claim that patients never recover. This may be true if by recovery we mean a complete restoration to the normal mental health, but if the patient be treated early, he will often be apparently restored to normal, the confusion will disappear, the disorientation vanishes, and he becomes clear and intelligent. If, however, there be any fresh indulgence in alcohol, as is usually the case, the symptoms will speedily reappear, and the patient may return to the hospital in a graver mental state, which may end fatally with the symptoms of "wet brain."

Treatment.—In the beginning of the disease and in all cases of delirium and excitement, the first requisite is rest in bed. If the patient be restless, and unwilling to stay in bed, he may sometimes be appeased and kept quieter by the persuasion of the nurses, one or more of whom should be in constant attendance. Very excited and violent cases may be relieved by being kept in a warm bath for hours at a time; in milder cases a warm pack may be of service, but the mechanical restraint of the pack is apt to be disturbing. Other forms of restraint, the camisole and tying in bed, are to be condemned. With insufficient or inefficient nursing they may be required, but their use means greater mental disturbance and an increased death rate. The second requisite is an abundant supply of food. In many cases liquid food, milk, eggs, strong broths, etc., are preferable, and in alcoholic cases especially they should be given hot. Restraint by sedative drugs is undesirable, but in cases of delirium tremens drugs may be used to procure sleep. Paraldehyd, in drachm doses (gm. 4), bromids in doses of 20 to 40 gr. (gm. 1 to 3), or hyoscin subcutaneously, $\frac{1}{100}$ gr. (gm. 0.0005), give the best results. In alcoholic cases it was formerly recommended to continue giving alcohol, lest the sudden withdrawal should precipitate an attack of delirium tremens. This is an outworn superstition, and no treatment is more to be condemned than to treat a case of poisoning by giving more poison. The result, as I have repeatedly seen, is simply to convert a comparatively mild case of alcoholic insanity into a more severe and chronic type, or even to hasten a fatal termination. If there be any indication of cardiac weakness it may be met by good-sized doses of digitalis, ♏ x to xl (gm. 1 to 3), nux vomica, aromatic spirits of ammonia, carbonate of ammonia, or camphor. In alcoholic cases with gastric disturbances, carminatives (capsicum, ginger, etc.) are of some service. In addition to this, we should promote free elimination by the intestines and kidneys. After the acute stage of the disease is past, the patient may be allowed gradually to get about. In this stage, residence in the country, where he can be isolated from all visitors, with competent nurses, abundance of fresh air and good food, are the essentials. He should not be permitted to exercise much or to have any excitement, and much rest in bed is still essential. Careful oversight must be constantly employed. Massage, mild gymnastics, and graduated mental occupation, reading, games, various forms of handicraft, gardening, etc., are all of benefit in the later stages. Mental occupation under careful restrictions is of especial importance. Tonics and other forms of symptomatic treatment may, of course, be employed. It is obvious, however, that such treatment is expensive, so that many cases will have to be sent to a hospital for the insane.

CHAPTER II

DEMENTIA PRÆCOX

(Primary Dementia. Hebephrenia. Catatonia. Adolescent Insanity. Schizophrenia. Dementia sejunctiva. Sensory Phrenosis)

Ætiology.—The cause of dementia præcox is still very obscure, if not wholly unknown. Heredity is given the first place, and some form of neuropathic disorder may be found in the antecedents in from sixty to seventy per cent of the cases. There may even be a similar heredity—that is, the occurrence of the disease itself in the antecedents, especially on the maternal side, and several members of the family may be afflicted with the disease. Dementia præcox, indeed, seems to be one of the few diseases of the nervous system, with hysteria migraine and maniacal depressive insanity, in which similar heredity can be maintained with any force. As has already been said (see the chapter on Epilepsy), the mere fact of nervous disease occurring in the antecedents is not necessarily a proof of any special predisposition to nervous disease in the individual, nor even an indication thereof, unless the incidence of such nervous disease be far more frequent than the average. It is possible that in dementia præcox there may be a congenital tendency of certain systems of neurons, especially those having to do with processes of association in the cerebral cortex, to fail prematurely—the so-called abiotrophy of Gowers—just as certain systems of neurons in the cord are supposed to fail prematurely in various family affections, such as Friedreich's ataxia. In some cases we can discover that the victim of dementia præcox was always peculiar, nervous, perhaps mentally below the average, and physically weak; but, on the other hand, it may occur in persons who have been previously in good physical health, who show none of the so-called stigmata of degeneracy, and who previously had distinguished themselves intellectually in school or college. A considerable percentage of the cases seems to occur in persons of a reticent, secretive, "shut-in" type. Age has an important influence, inasmuch as in about sixty per cent of the cases the disease makes its first appearance before the age of twenty-five, and usually in the period between fifteen and twenty-five. The disease may occur, however, in the first decade of life, and even at the age of fifty. The sexes are about equally affected, although in the hebephrenic type more males are affected, and in the other types more females. The influence of infection and intoxication has been variously estimated, but it apparently is of considerable importance in the genesis of the disease. Emotional shock, injury, and imprisonment are also of some influence. The injurious influence of masturbation was formerly thought to be so potent a factor in causing the disease that many cases were classed as "masturbation insanity," and this belief still prevails among the laity. Masturbation, however, is usually to be regarded as one of the common symptoms of the disease in its incipient stages, indicating defective mental control, rather than as a cause, and careful inquiry will usually show that other symptoms of the disorder have been present at the same time with or even before the indulgence in the habit.

Symptoms and Course.—The disease usually begins with neurasthenic symptoms. The patient complains of headache, dizziness, disturbed sleep or loss of sleep, bad dreams, loss of appetite, indigestion, and general weakness; he may be melancholy, anxious, apprehensive, conscious that he is not well and dreading the onset of some disease, especially of insanity. He is often nervous, irritable, restless, unable to concentrate his attention on any subject, like the neurasthenic; but, in addition to all this, there is a noticeable change in his mental state which differs entirely from that of the neurasthenic. The neurasthenic complains of mental inadequacy, of inability to make any physical effort, to read, to concentrate his thoughts, or to do any mental work, all of which is perfectly true to a certain degree, but at the same time he can do all these things in a perfectly normal fashion for a brief period; he is not wholly incapable, but merely incapable of prolonged physical or mental effort, and is much more easily exhausted by any slight exertion. With the victim of incipient dementia præcox, on the other hand, an actual mental defect is soon observable. He is found to be unable to do his regular work as well as he did before, to be forgetful, distraught, and to make unaccountable and often foolish mistakes. He takes up new occupations with considerable enthusiasm, yet without a just appreciation of his own capacity, and gives them up in a short time or proves himself wholly unfit to continue in them. With his depression and feeling of mental disability he begins to question about all sorts of metaphysical problems, often without any adequate knowledge of the subject upon which he ponders. He accuses himself of incompetence or sin, he thinks his life is useless, that he is not in the position which he ought to hold either socially or intellectually, that he is ill-treated, and that people oppose him and work to keep him from attaining his rightful high position. He shows more or less confusion and forgetfulness; he may read, but he fails to comprehend what he reads. He talks about a change in his nature, of a feeling of inadequacy and insufficiency, of a disgust for life, and of the hollowness of all about him. His mental processes suffer, he no longer reasons logically, his attention is readily diverted, and his power of work is much diminished.

With all this there is a curious childishness which is never met with in the ordinary neurasthenic. The patient reveals all sorts of romantic, visionary ideas as to his abilities, his plans and his future career, which are manifestly at variance with his actual position and attainments, and which are not corrected by any intelligent observation and judgment. He may advance some idea of becoming a Napoleon of finance, a great actor, a profound philosopher or a brilliant mathematician, without regard to the necessary preliminary qualifications or training for such a career. His notions are silly, extravagant, variable, and confused. The ideas of the neurasthenic, on the other hand, are always clear, rational, and in accordance with his mental development.

This initial mental change is usually soon followed by the development of hallucinations, of which the hallucinations of hearing are apt to be the commonest. Delusions soon arise, chiefly of a persecutory character or else relating to the commitment of sin. These are usually present in all forms of the disease, but they are often of short duration and unsystematized. They may, however, in the paranoid form, be systematized and continue throughout the disease. Hypochondriacal delusions are common, yet they are often of a childish and absurd nature. The patient may insist, for example, that he is so weak that he cannot lift his arm, yet, while he is arguing to that effect, he may hold a weight with his arm extended, and, when told what he is doing, will have no reason to advance to explain the discrepancy between his actions and his statements. If he argue that he has committed the unpardonable sin and is doomed forever to hell, he does not show the distress that accompanies this delusion in melancholic conditions, nor does he manifest the fear shown by the paranoiac at the persecutions and plots of the Czar, the Freemasons, or the Mafia. In all his delusions may be traced the same inconsequential, childish, affected attitude.

When the disease is fully developed the mental faculties show various characteristic modifications. The consciousness is usually clear, although at times there may be some cloudiness and confusion. The memory, too, is not at first very noticeably impaired, and even in the later stages there is often a surprising retentiveness of former events and things learned before the onset of the disease. Except in conditions of stupor or marked excitement the patient rarely shows disorientation; he retains a fair idea of time and place, recognizes his associates, and may sometimes call them by name after years of silence. He has fairly good perception, and may even be able to receive and retain new ideas, but the power of continuous attention is very markedly impaired. It is difficult to induce the patient to follow any consecutive train of thought, yet he often develops a special, limited, almost stereotyped trend of thought on his own account. He loses interest in everything about him, takes no enjoyment in the pleasures of life, is indifferent to the fate of his family or his friends (affective dementia), although he is often depressed and sometimes emotional. The failure of judgment has already been alluded to, and is one of the marked features of the disease. There is, too, a marked lack of volition; the patient often seems quite incapable of independent activity, and, if left to himself, will sit for hours without moving or speaking. There is a slowing of the mental processes, the reaction time is unduly prolonged—that is, it takes a much longer time than normal for the patient to respond by any action to a given signal; his answers to questions, if he answer at all, are given only after a protracted interval; there is an inhibition of all mental processes.

The influence of this mental condition upon the conduct and actions of the patient is soon evident. One of the early symptoms is a certain stolidity of conduct, a general passive state in which the patient is indisposed to make any effort, which is due in part to his general lack of interest in his surroundings. At times this may increase to a positive resistance to any request or to any attempt to make him do anything or move from one spot—negativism. This may go so far as to cause the patient to remain in some fixed attitude for days at a time, or to exhibit the phenomena of catalepsy (*q. v.*). In the more severe cases in the asylums we often see patients who, if permitted, spend most of their time sitting, standing, or lying in some peculiar and apparently uncomfortable position, which they may have taken originally in response to some delusion, but the reason for which has faded from their minds. In the earlier stages, especially, we observe these same stereotyped attitudes and various mannerisms of action and speech which are often assumed as a result of some delusion or simply as a dramatic form of expressing their ideas. Many patients have a foolish, romantic, or dramatic manner; they imagine that certain attitudes or actions are symbolic of some idea; they wish to dress in a peculiar fashion, to make certain signs as a symbol, to speak in a certain affected way, to adopt a special manner of entering a room, of taking a seat, or of shaking hands. Not only in the manner of speaking, but in the contents of speech as well, the patient may show similar mannerisms. He may answer in rhyme or assonance, or use strange or original words (neologisms), or frequently interject some word or phrase into his conversation. Some patients are fond of writing, and will fill reams of paper with high-flown, sentimental, romantic, and incoherent sentences, often accentuated by an absurd use of italics, capitals, spacing, the arrangement of words on the page, or perhaps by pictures. Sometimes the patient is easily influenced by suggestion or by imitation. He will perform certain automatic acts, or repeat words or acts that are done in his presence (echolalia, echopraxia).

Although the patient is often apathetic, indifferent, and takes no interest in the world, yet this is not always the case. Very often, especially in the earlier stages, there is a considerable increase of sexual desire, which, with the deficient mental control, may often lead to scandal. It is probable that, in a part, at least, of the cases where insanity is said to have followed seduction, the seduction has occurred in the early stage of dementia præcox. This same increase of sexual desire gives rise to the very frequent and often excessive masturbation which was once thought to be an important cause of the disease. In the early stages, in particular, where there is much depression, anxiety, and the fear of insanity, attempts at suicide are not infrequent. In other cases there may be senseless outbursts of violence, or single impulsive, violent acts.

The general trend in all these cases is toward a mental deterioration leading to dementia. This dementia differs, however, from that seen in general paralysis or in senile cases. Although the patient may have sunk into an automatic state, where he does not speak and remains in one attitude for hours, there will sometimes be a flash of intelligence indicating that he is still conscious of his environment, and a shrewd attendant can sometimes transform an apparently stupid dement into a moderately efficient worker.

The disease may be attended with various physical symptoms. Circulatory symptoms are quite common; the heart may beat slow or fast, and is often weak and irregular; there is often cyanosis or coldness of the extremities, sometimes a circumscribed œdema, dermatographism, profuse sweating, or dryness of the skin. There may be some rise of temperature at the onset, but it often escapes notice; later on, however, the temperature may be below normal. The nutrition is markedly impaired in the early stages, the appetite is lost, there are various digestive symptoms, salivation, a coated tongue, flatulence, dyspepsia, constipation, etc. Later on there may be an increase in weight, when the digestion is once more reëstablished. Menstruation may become irregular or wholly cease for several months. The pupils are often dilated and unequal, and sometimes rather sluggish in their reaction. There may be some difficulty in looking fixedly at any object or in following a moving object persistently. Corneal insensibility, contraction of the visual field, and slight degrees of optic neuritis have been described, but they are not very common. The thyroid gland is sometimes enlarged. The urine may be scanty, and show some deficit in urea and excess of chlorids; albuminuria is rare. The knee jerks and other tendon reflexes are often exaggerated, especially at the onset, but they may be diminished. The mechanical muscular irritability is sometimes increased. Disturbances of sensation, especially hemianæsthesia, are not uncommon. Various attacks of faintness, hiccough, localized spasm, rigidity, contracture, choreiform movements, or actual convulsions, are sometimes observed.

The course of the disease may vary. Some cases have attacks of depression lasting a few months with the milder symptoms above described, and recover, remaining well for a number of years, when a second attack with more pronounced symptoms may develop. Occasionally there may be periods of depression and excitement, resembling the circular type of maniacal-depressive insanity. Many cases, after the initial period of the disease is past, remain in an unchanged condition for the rest of their lives, either in a paranoid or somewhat demented state. In other cases there is a gradual increase in the dementia, sometimes due to the conditions under which they live ("asylum dementia") rather than to an actual extension of the morbid process in the brain. Sometimes there is a very rapid development of dementia, which may reach a marked degree in a few weeks.

Three clinical types of dementia præcox are ordinarily described—hebephrenia, catatonia, and paranoid dementia—but mixed types may occur, and also a form of simple dementia (heboidophrenia).

HEBEPHRENIA.—This type of the disease usually begins with neurasthenic symptoms; the patient complains of headache, dizziness, and inability to sleep. The appetite and digestion are affected, and nutrition is considerably impaired. The disposition seems changed; if the patient be of the reticent, secretive type, the normal peculiarities are exaggerated. Other patients also become shy, reticent, and self-absorbed. They neglect their work, become forgetful and distracted, and cannot do their work as well as before. They are dreamy, moody, and brooding. If disturbed they are irritable, and are often restless, depressed, and anxious. If away from home they show marked homesickness. The depression and restlessness may lead them to wander away, or to make attempts at suicide. Sexual excitement, especially in the form of excessive masturbation, is common. Hallucinations and delusions develop, but they are usually inconstant, unstable, and of short duration. The patients become dull and indifferent, the understanding and intelligence are impaired, and they are incapable of mental work, or, indeed, of any physical work requiring persistent attention. They are conscious that something is the matter, and often fear insanity, yet there is often the childish, affected, inconsequential way of talking; the romantic, visionary, dreamy disposition; the lack of any just appreciation of the relation between their dreams and the actual conditions under which they live. The memory, orientation, and perception are often not much affected, but there may be some confusion and lack of clear conception. They often show a marked emotional defect, care little for their friends or family, and fail wholly to appreciate the care and privations to which the family may be exposed by their illness. They become extravagant in action, or even violent, and commit various senseless and peculiar acts. The tendency is toward a steady increase in the dementia, but many cases remain stationary after the first attack, and some recover.

CATATONIA.—The catatonic form also begins with a neurasthenic stage, but the symptoms of mental depression soon become prominent. The patient complains of headache, loss of sleep, poor appetite, dullness and difficulty in thinking, and gives up his usual occupations. He shows a greater tendency to seclude himself, and often remains in bed. Hallucinations and delusions soon make their appearance. The latter frequently assume a religious character, the patient shows a greater fondness for religious observances, prays without ceasing, is oppressed by a conviction of sin, accuses himself of extravagant and unusual crimes, often of an absurd and foolish nature, believes that he has committed the unpardonable sin, sees hell opened to receive him, and the like. Persecutory delusions are also not uncommon. These delusions often exhibit that romantic, childish, and unreasonable character already mentioned. The patient may argue that he is no longer alive, that he is non-existent, has no internal organs, or has destroyed them by his sins. Later on these delusions may assume a more exalted character, and, in consequence, he becomes more exhilarated. The disease is apt to assume either a stuporous or an excited form. In the stuporous form the patient grows silent, dull, and somewhat confused. He has no interest in his surroundings, and soon assumes some stereotyped position, in which he will remain for hours. He shows marked negativism, resisting all attempts which may be made to get him to move about, to eat, or to attend to the calls of nature. He answers in monosyllables or not at all. He refuses food, and may even retain his urine or feces for a long time, finally voiding them wherever he may be. All his actions and mental processes are remarkably slow. If he eats, he may spend hours at a meal. In the excited form there is more unrest, the patient moves about restlessly, and often talks unceasingly (verbigeration, logorrhea). The speech may be confused and disconnected, with the interjection of irrelevant words, the use of newly invented words, rhyme, etc., which is sometimes spoken of as a "word salad." The actions show the affected mannerisms already mentioned, and tend to become stereotyped and few in number. There may be cataleptic states, and the patients are often suggestible, performing various semi-automatic acts as a result of suggestion, either by direct command or by imitation (echopraxia). Sudden impulsive and violent acts may be performed. The memory for past events may be fairly good, and the patient may rather readily learn to recognize his attendants and surroundings, and at times show some flashes of intelligence, but there is much more mental confusion and defective orientation than in the hebephrenic form of the disease.

Paranoid Dementia.—The paranoid form may also show similar symptoms at the onset, but the characteristic feature is the greater prominence and the persistence of hallucinations and delusions. The delusions often become systematized, and remain fixed for a long period. Various catatonic symptoms may show themselves in the course of the disease—stupor, excitement, peculiarities of speech and conduct, negativism, confusion, and the like, but they are less pronounced than in the catatonic form. The consciousness is usually clear, the patient is fairly well oriented, recognizes his family, and has a fair memory of the past. At times he will insist that the people about him are really celebrated persons, often persons associated with his delusions, but now and then he will call one of them by his real name. The delusions, especially at the first, are apt to be of a persecutory character; the patient is influenced by hypnotism, telepathy, the telephone or wireless telegraphy, people enter his room, read his thoughts, deprive him of his vitality, cut him open and remove his organs, inject poisons into him by night, and the like. He is disturbed by these persecutions, anxious, and depressed. Later on his disposition may show a change. These persecutions are inflicted by some distinguished person or organization—the Mikado, the Catholic Church, the labor unions, because he is himself a distinguished person hostile to them, a king, an emperor, the Son of God, etc. Thus his mood may change from depression to expansion, and his delusions assume a grandiose character. His conduct is influenced by these delusions, which may lead him to perform many peculiar actions, or to show violence to those about him. The delusions often show the fantastic, childish character above mentioned. The mental faculties at first are not greatly affected; the patient may be able to pay fair attention to matters in hand, to understand fairly well, to think logically, but the train of thought is influenced by the delusions—he has defective insight into his condition, his attention is readily distracted, and he is incapable of any steady work. With this there is often a marked mental deterioration, with increasing confusion and dementia, but some cases remain in a paranoid state for years.

The simple dementia (*heboidophrenia*) differs from hebephrenia merely in the fact that here is a more or less rapid deterioration of the mental faculties, with faint delusions and hallucinations, and less of the affectations and mannerisms. In the descriptions given above it will be evident that the characteristic features of one type—the depression, stereotyped attitudes, mannerisms, and verbigerations of catatonia, or the delusions and hallucinations of the paranoid form—occur in other types, and that the difference is merely in the degree in which one or the other set of symptoms develop. It will therefore be evident that mixed types and transitional forms may occur, all of which may show the mental deterioration which is the chief characteristic of the disease.

Pathology and Nature of the Disease.—The pathology of dementia præcox is still obscure, but much light has been thrown upon the subject by the researches of Alzheimer, Sioli, Klippel and Lhermitte and Southard. The cells in the brain cortex show distinct changes, which are, however, similar to those found in toxic delirium and in severe bodily disease. There is an atrophy of the nerve cells of the deep cortical layers and disappearance of the interconnecting processes, with fibrillary gliosis in the subpial zone, the deep layers of the cortex, and even in the white matter, with some atrophy of the convolutions and the presence of amœboid neuroglia cells, especially in the vicinity of the nerve cells—satellitosis. The foci of gliosis are sometimes to be felt in the fresh brain. The projection system—that is, the cells and fibers belonging to the various sensory and motor systems—is little if at all affected, as is shown clinically by the absence of any paralysis or anæsthesia, and the morbid process is believed to cause a progressive atrophy of the association neurons. There is some reason to suppose that in the paranoid form these changes are more marked in the prefrontal region, and that in the catatonic form they are more marked in the parietal regions and in the cerebellum (Southard). These pathological changes may vary very greatly both in their intensity and in their distribution, the variation corresponding probably to the intensity and the clinical type of the disease. Some of the changes, furthermore, are very similar to those observed in the various cases of confusional insanity of toxic or infectious origin, and a number of authorities are disposed to think that toxic processes, often endogenous, play a very important part in the ætiology of dementia præcox.

The symptoms of the disease may fairly be attributed to changes in the processes of mental association rather than to any actual failure of perception, to lesions of the association system rather than of the projection system. Some of the symptoms, which give a very characteristic coloring to the clinical picture, may perhaps be explained in another way. A very considerable number of cases develop somewhere about the age of puberty, or, at any rate, during the period of adolescence. Recent studies of puberty and adolescence (Marro, Hall) have emphasized certain mental characteristics of this period—a tendency to reverie, romantic imaginings, religious and erotic fancies, dreams of future achievements uncontrolled by any just appreciation of the dreamer's abilities, idealistic aims, and the like, often associated with mysticism, sentimentality, and even downright foolishness. These tendencies are much greater in the delicate, weak, shy, reticent person of the "shut-in" type than in the vigorous athlete. If such a person during the period of adolescence be affected by mental disease, these characteristics will assume a prominent place in the clinical picture. If the patient, in addition, be enfeebled from birth with an "invalid" brain bearing some hereditary taint, the disease may cause far more irreparable damage to the brain than it would to the brain of a healthy person, and thus give rise to a more or less rapidly developing dementia. It may be, therefore, that the distinction between confusional insanity and dementia præcox is due rather to the difference between the individuals affected, and also to the difference in the severity of the disease, rather than to any difference in the morbid process itself.

Diagnosis.—The important elements in diagnosis are the initial neurasthenic period, with marked depression, during the period of adolescence; the development of hallucinations and delusions with the peculiar, romantic foolishness above described; the appearance of negativism with affected mannerisms and stereotyped actions, and the more or less rapid onset of dementia. The distinction between the prodromal stage and simple neurasthenia has already been described. The differentiation from hysteria in the early stages is more difficult. The occurrence of some of the so-called stigmata of hysteria—hemianæsthesia, contracted visual fields, convulsive attacks, etc.—has already been mentioned, and has given rise to the suggestion that hysteria may at times coexist. The development of catatonic symptoms, the existence of profound apathy, the progressive dementia, the tendency to negativism, and the special childishness of thought and action will usually make the diagnosis possible. The cases that begin with marked melancholia, with remissions and phases of mild excitement, sometimes suggest melancholia or circular insanity. Here again the apathy, catatonia, negativism, and progressive dementia favor the diagnosis of dementia præcox. The distinction between dementia præcox and confusional insanity is often difficult. The diagnostic criteria are stated to be that in dementia præcox the onset is gradual, and there is no previous history of exhaustion or of toxic influences. Negativism, verbigeration, mutism, stereotyped attitudes, and catatonic states favor dementia præcox. Perception and orientation are fairly well preserved; confusion is less common, and emotional apathy is marked. In confusional insanity perception and orientation are much affected, and confusion is a striking feature. It may be said, however, that many authorities, especially in France and Italy, believe that the two affections are practically the same, dementia præcox being merely a more profound type of the disease, perhaps modified by its occurrence in adolescent and neuropathic individuals. The differential diagnosis between the paranoid form and true paranoia will be discussed later.

Prognosis.—The outlook is always grave, but the prognosis is regarded as somewhat more favorable in the catatonic form than in the hebephrenic, while the paranoid form rarely recovers. About eight per cent of the cases of hebephrenia and thirteen per cent of the cases of catatonia are said to recover. Cases of the circular type are rather more apt to recover, at least temporarily, but if the circular symptoms disappear without improvement in the mental conditions, recovery is not likely to occur. Cases of simple depression, with subjective sensations of mental inhibition, are also likely to recover temporarily, recurring with catatonic symptoms. The prognosis also seems somewhat more favorable in catatonic cases if the stupor or excitement be pronounced, or if there be anxiety and delirium. A considerable number of cases show so much improvement that they may be called cases of recovery with defect. They are able to return home, to prosecute various callings, and to take some place in the world, but they do not regain their former intellectual plane. They are not capable of as good work as before; they show some social and ethical deterioration, and may present traces of former delusions. A certain number of the milder cases that make a partial recovery are found among the criminals, prostitutes, and tramps. In cases that recover, the disease may recur after a period of several years. In the early stages of the disease, death may occur, usually as a result of intercurrent disease. Tuberculosis is very apt to develop, especially in stuporous and much-demented patients. Many cases, under proper treatment, may regain complete physical health, be trained to do simple work under supervision, and live for many years.

Treatment.—The treatment in the early stage is similar to that of confusional insanity—rest in bed, abundant feeding, and careful oversight. As attempts at suicide and sudden outbursts of violence are not uncommon, especial oversight is required, and most cases will need the restraint of a suitable hospital. On the hypothesis of a possible toxic influence in the ætiology, free elimination should be encouraged. Treatment directed to a possible thyroid origin (thyroidectomy, thyroid preparations) has been recommended, but without any recognized benefit. Many cases, after the first acute period of the disease is past, would do better outside an institution, where they could be under careful supervision to prevent any ethical lapses, yet where they could undertake some simple occupation, perhaps under oversight and direction, especially work out-of-doors or some manual occupation. Methodical instruction, such as is given to the feeble-minded, but adjusted to the individual case, has been of distinct benefit in reviving dormant mental powers and in enabling the patient to retain, if not to regain, some portion of his intelligence. If left to himself, as is so often the case in large institutions, the patient tends to remain in some stereotyped attitude and become more and more demented, which can often be prevented by proper training.

CHAPTER III

MANIA. MELANCHOLIA

(*Maniacal-depressive Insanity. Recurrent Mania or Melancholia. Lypemania. Periodical Insanity. Alternating Insanity. Insanity of Double Form. Circular Insanity*)

Ætiology.—A neuropathic heredity may be found in about eighty per cent of all cases of maniacal depressive insanity, and it is one of the few diseases of the nervous system in which a similar heredity seems to be comparatively frequent and of some ætiological significance. Several cases may occur in the same family. About two thirds of the cases occur in women. About the same proportion of cases develop before the twenty-fifth year, and cases have been observed even as early as the age of ten. Other factors—infection, intoxication, worry, and emotional strain—may have an influence in provoking an individual attack. In about one fourth of the cases an attack of some physical disease has been found to precede the first attack, but the incidence of physical disease was found to diminish in the second and third attacks. Abuse of alcohol is of some importance, both as an hereditary factor and as provoking the onset of the disease.

Symptoms. *Mania.*—Mania may come on suddenly, possibly as a sequel of some emotional excitement, but more commonly there are some prodromal symptoms, depression, malaise, a change in disposition, and a certain unfitness for the usual duties of life. There are not infrequently loss of appetite and disturbances of digestion. Soon after this the patient becomes restless, excitable, brusque in manner, exhilarated and talkative. He seems quicker in speech and action, mentally more alert and brilliant, is lively, cheerful, and fond of joking; he even regards life and all that is done around him as a jest. He talks much more volubly than usual, becomes very prolix, and is apt to disregard the ordinary conventions in his talk, and to gesticulate more freely. He is much more restless, is busy about many things, yet really accomplishes little. To the casual observer he may seem unusually brilliant, or, sometimes, as if he were unduly exhilarated by alcohol, but on closer investigation it will be observed that he has lost some of his ability to carry out any logical train of thought or any continuous series of acts to their conclusion; he is easily distracted, turns suddenly from one thing to another, and acts impulsively without judgment or consideration. There is a tendency to behave peculiarly, to disregard the ordinary rules of conduct, to make rude jests, to treat people roughly, or perhaps to be guilty of more serious lapses; to indulge immoderately in alcohol, to spend money recklessly and foolishly, or to frequent brothels or make himself conspicuous with prostitutes. With the exhilaration there is an undue conceit of his own abilities; he develops grandiose schemes, and plunges recklessly into speculation. There is no disturbance of consciousness—the patient is well oriented, the perception and attention are quicker than normal, but the attention is readily distracted. The memory is good, but the memory of recent events is sometimes colored by the exhilaration, so that the patient's statements become untrustworthy. In spite of the exhilaration the patient is unduly irritable, and, if opposed, may grow very angry and sometimes violent. The increased loquacity and the tendency to rush from one subject to another suggest an unusual mental activity, an actual "flight of ideas." It will be found, however, that the reaction time is actually prolonged, that the mental activity, in the nature of continuous thought, of careful reasoning and sound judgment, is actually lessened, and that the apparent brilliancy is due to the lack of restraint, the excessive talking, and the tendency to rush from one subject to another without the control of the judgment. There is also less actual wit in what is said. The wit is more often of a simple character—puns or personal remarks which are often amusing from their bluntness, yet which refer to obvious peculiarities that a well-bred person would not mention. The appetite may become increased, but there is still sleeplessness. In spite of all the activity, however, there is little or no feeling of fatigue.

This comparatively mild type of the disease, known as simple mania or hypomania, may persist throughout the attack, or it may develop more or less rapidly into the more pronounced form. The patient becomes much more agitated, talks incessantly, and is constantly in motion. The attention is much more readily distracted, the speech becomes incoherent, often a flood of words associated by rhyme or assonance; the patient may write, but the writing is hurried and disordered, and shows the same incoherence of ideas. Mimetic movements are more pronounced; the patient gesticulates freely and in extraordinary fashion. He is not content with simply talking, but sings or shouts until he is hoarse. There is a tendency to stereotyped actions and verbigeration. He is extremely excitable, and becomes violent and even furious if opposed. There may be considerable confusion, orientation becomes defective, consciousness may be clouded, and occasionally hallucinations and delusions of a fleeting type may develop. At times he may be depressed and tearful, instead of cheerful and exhilarated, but the depression is usually of short duration. There is an apparent lack of sensibility to heat and cold, to hunger and thirst, and to fatigue. Patients will strip off their clothing in winter and apparently not mind the cold, or expose themselves to the sun on the hottest day in summer. They will shout and rave all day and most of the night without any diminution of their energy, without sleep and apparently without need of rest. The sexual desire is often increased. Women often expose themselves without shame, use the most obscene and profane language, and make advances to any man who approaches. In the more extreme degrees we have the familiar picture of the raving maniac, agitated, incoherent, noisy, violent and furious, destroying not only his own clothing, but everything about him.

The physical symptoms are not very pronounced. The digestion is often impaired, the tongue coated, the appetite variable, and the bowels constipated. The pulse is frequently rapid, the circulation good, and the blood pressure not remarkable. The reflexes are often lively at first, but diminished later. The urine is apt to be scanty, and sometimes shows an excess of urea and of phosphates. Menstruation often ceases during the attack, or becomes scanty and irregular.

Melancholia.—Melancholia may also begin suddenly after some emotional strain, but more frequently there is a prodromal stage in which the patient seems more impressionable and depressed, complains of headache, sleeplessness, and loss of appetite, has a disturbed digestion, and loses flesh. The characteristic feature is a profound sadness, out of proportion to the actual conditions of life, which, of course, may be of a depressing character. With a distaste for everything and a general indifference, there is a feeling of absolute impotence, of complete inability to think, will, or act. All thought seems difficult, and there seems to be an inhibition of all mental activity. When there is any mental action it is extremely slow. The patient talks little, if at all, spontaneously, and the answers to questions are made in a low tone, often in monosyllables or in as few words as possible, and only after a considerable interval. The patient has no interest in life, can find pleasure in nothing, all seems dark and hopeless, and he is a prey to all manner of gloomy thoughts, and broods and worries over trivial matters. At the same time he is mentally clear and well oriented, the memory is good, and the judgment and understanding are normal except as they are influenced by the slowness of thought and the depression. The attention can only with difficulty be fixed, and is easily distracted, and all associations are much slower. Everything seems to be an effort, and the patient loses his power of action. He neglects his work, his ordinary duties, and even his personal needs. He complains of being ill, of various distressing cerebral symptoms, pain, discomfort, light-headedness, pressure in the head, etc. The appetite is very poor—he may refuse food absolutely; the tongue is coated, the saliva scanty and viscid, the breath offensive, and the bowels constipated. The patient loses weight. He sleeps poorly. The expression is mournful, the eyes dull, the skin very dry, faded, and wrinkled. There is often anæmia, the number of red corpuscles is diminished, and there is a lessened amount of hemoglobin. The blood is said to be less toxic, but there is increased toxicity of the urine. Respiration is slow and superficial, and the patient may complain of suffocation and palpitation. The reflexes are diminished. Menstruation may wholly cease, or at least become scanty and irregular. Sexual desire is almost wholly lost. There is often distinct analgesia.

In the milder forms of the condition, which is sometimes termed simple melancholia, the patient may, by a great effort, go about and do his ordinary duties, although less efficiently. In the severer forms, which may develop rapidly upon the condition just described, there is much more mental disturbance. Actual hallucinations are comparatively rare, but delusions often develop. These may take the form of delusions of culpability; the patient feels that he has committed some grave sin, that he has been unchaste in thought or deed, that he has committed or thought of committing some obscene act in a church, that he has sinned against the Holy Ghost, and thus committed the unpardonable sin. In other cases the patient has the idea that he is coming to want, that he is destitute, that he and his family must starve or go to the poorhouse, and no presentation of facts can shatter his conviction. Another less common form of delusion refers to the patient's physical condition, and is of a profoundly hypochondriacal nature; he has some incurable disease, such as a cancer, his bowels are closing up or are paralyzed, his sexual organs are no longer efficient, and the like. Sometimes he is disposed to deny the existence of his organs, the objects about him, etc. (delusion of negation). Delusions of persecution are rare. These delusions are never primary, but develop secondarily after the patient has become depressed; they are painful in character, monotonous, and have something of the nature of obsessions. The patient himself is humble, and always accuses himself as the author of his misfortune, and his explanations of the conditions are retrospective, referring to some act committed years before. Furthermore, he is constantly expecting evil results in the future; he is going to be killed or doomed. The mental depression increases, attempts at suicide are frequent, and the patient may refuse food altogether.

The severe type of melancholia is apt to take one of two forms—the stuporous or the agitated. In the stuporous form (*melancholia attonita*) the patient becomes weak, assumes a fixed position with an expression of profound distress, seems in terror when spoken to or made to move, refuses food, and remains for hours in a position suggesting a catatonic dement; the facial expression alone is different. In some cases this mutism is the result of terrifying delusions. In the agitated form (*melancholia agitata*) the patient is restless, moaning, groaning, and crying. He wrings his hands, bites his nails, picks the skin off his face, tears out his hair, and is seemingly oblivious to any pain from such actions, due perhaps to analgesia. At times he may mutilate himself to an even greater extent. He becomes filthy, and will daub himself with feces, or even eat his own feces and drink his urine. In both conditions the physical symptoms above mentioned increase in severity, the physical condition becomes worse, the patient becomes much emaciated, and may succumb to starvation or to some septic state resulting from self-inflicted injuries.

In some cases melancholia may not develop until later in life, between the ages of fifty and sixty—the "involutional melancholia" of Kraepelin. These cases are apt to assume the delusional type, but the delusions are not as well developed, and there is less of the psychomotor retardation, or slowing of all mental and physical activity.

Course.—A considerable percentage of the cases of mania and melancholia recover in a period of from two or three months to a year, but some cases become chronic. A patient, however, who in youth has had one attack of mania or melancholia very often, although not inevitably, has other attacks later in life. These attacks may vary greatly in duration, from a week or two to a year or more, and they may come at varying intervals, from a few months to many years. As the attacks recur, the tendency is for the sane interval to grow shorter. Sometimes the attacks come on at very definite intervals, so that the patient can prepare for them and go to a sanitarium to be ready for them. The manner of recurrence may also vary. A patient may have recurrent mania, with maniacal attacks recurring regularly, or recurrent melancholia. In the irregular types an attack of melancholia may occasionally replace the attack of mania, or *vice versa.* In alternating insanity we find attacks of mania and of melancholia in regular alternation, each attack separated from the next by a sane interval. In the "insanity of double form" the attack of mania passes at once into melancholia, or *vice versa,* and then comes a sane interval before the two attacks recur. In continuous circular insanity the patient passes directly and suddenly from an attack of mania into an attack of melancholia, and then becomes maniacal again. Mixed types also occur, and sometimes during a period of melancholia there may be spells of excitement of a maniacal type, or a maniacal patient may have transitory spells of depression.

Pathology and Nature of the Disease.—Nothing definite is known as to the pathology of the disease. Changes of some sort are usually found in the brain, but they are often to be explained by other conditions—arteriosclerosis, intercurrent disease, etc. Some writers have attempted to explain the occurrence of the disease by vasomotor changes, and have thought that there were always changes in Helweg's triangle in the medulla which would affect the vasomotor centers there. The clinical evidence of vasomotor disturbance is slight, and recent observers have failed to find the alleged changes in Helweg's triangle. Kraepelin has maintained that all cases of mania and melancholia, except the "involutional melancholia," are manifestations of one disease, to which he has given the name of "maniacal-depressive insanity," of which circular insanity would be the ultimate type. Some writers, however, maintain that there is clinically a distinction between a single attack of mania or melancholia and the attack of mania or melancholia that occurs in circular insanity. The single attack is said to come on after some definite cause—emotional strain, physical disease, etc.—and to be ushered in by a prodromal stage. It is also maintained that there is not a fatal tendency to recurrence, such as Kraepelin's theory would imply. At present, at any rate, we are at a loss to determine the nature of the affection, and the reason for its recurrence in the various forms.

Prognosis.—The prognosis of the individual attack of mania is comparatively good; rather more than half the patients recover. The prognosis in melancholia is not quite as good, especially in the melancholia that develops later in life. The prognosis for life, too, is distinctly worse in melancholia, from the dangers of suicide, inanition, and sepsis. If the disease show a distinct tendency to recurrence, especially if it assume the circular type, the outlook is, of course, unfavorable, especially if the sane intervals be of short duration. In some cases, however, when the sane intervals are long, the patient may lead a fairly active and useful life during the interval. In a good many cases there is a tendency, after repeated attacks, to the development of dementia, but it is possible that such cases are really the circular or recurrent type of dementia præcox, or that the dementia is due to the development of organic brain disease of arteriosclerotic origin.

Diagnosis.—The diagnosis is easy if there be frequent recurrences or if the disease assume the circular type. True circular insanity, however, is a rare disease. The diagnosis of a first attack is often very difficult. The cases of stuporous melancholia may readily be mistaken for the catatonic form of dementia præcox. In dementia præcox, hallucinations and delusions are apt to be early symptoms, there is often less actual sadness and emotional disturbance, and the negativism differs somewhat from the psychomotor inhibition. The patient may not move until he is touched, and he may then make some definite movement; the melancholic patient may make several abortive attempts to execute a spoken command even before he is touched. The development of stereotyped attitudes is rather in favor of catatonia, as is also the appearance of dementia. Many cases of confusional insanity and dementia præcox in a state of excitement have in the past been regarded as mania. The existence of marked confusion in the quieter periods, the lesser flightiness of ideas, the history of infection or intoxication, and the early appearance of delusions and hallucinations are in favor of the diagnosis of confusional insanity; the mannerisms, apathy, and progressive dementia are in favor of dementia præcox. General paralytics may at times show profound depression or become maniacal. The diagnosis can usually be made by the discovery of the physical symptoms of general paralysis, the pupils, the alteration of the reflexes, the disturbances in speech and handwriting. In cases of doubt the examination of the cerebro-spinal fluid obtained by lumbar puncture will be decisive.

Treatment.—The treatment of the milder forms of mania is often very difficult. The patient's exhilaration, lack of judgment and application, and the increased sexual desire may lead to very serious social disaster, yet the mental aberration is often insufficient to convince the family or the authorities of the need of restraint; strict oversight and control, if not actual restraint, are, however, often essential. The patient's own sense of well-being, too, is so great that he will not voluntarily submit to restraint or consult the physician. If he can be made to submit to treatment he should be carefully watched, given exercise or manual work in the open air to relieve to some degree his motor restlessness, the diet should be carefully regulated so that he may take a considerable amount of simple and nutritious food with plenty of fluid, but nothing of a stimulating character. Careful attention should be given to secure free elimination by the kidneys and intestines. The patients are often too restless to admit of bed treatment, but they should be induced to spend as much time in bed as possible. The use of continuous warm baths will be of distinct benefit in quieting the restlessness, and in the more violent cases such a bath is of great help. Actual physical restraint is usually an irritant, and often increases the excitement and violence, but a patient may be kept fairly quiet for hours in the bath, if he be not actually restrained, and be occasionally permitted to get out of the tub. Sedative drugs, of which hyoscin is the most frequently recommended, are to be avoided. In the more violent forms the patient will need the restraint of a suitable hospital.

Melancholia is one of the most dangerous forms of mental disease. Not only is there great danger of suicide, but the patient often attempts to involve others in his own destruction, and may murder his entire family. For this reason the most careful and constant oversight is often necessary. If the patient be aware of this, it may prove irritating, and perhaps exert unfavorable suggestions, so that in the milder cases it is sometimes advisable to make the oversight less obvious, even if the risks be thereby increased. Melancholic patients in the earlier stages will often consent to put themselves under treatment in some sanitarium. One of the first requisites in treatment is abundant feeding. This is often difficult, and sometimes the patient absolutely refuses to eat. In such cases there should be little delay in feeding by means of the stomach tube. This may require the aid of several attendants, so that it is usually advisable to have the patient in a suitable hospital. The tube is more easily inserted through the nose, care being taken to avoid entering the larynx. A slight gurgling may be perceived when the tube reaches the stomach. In this way the patient may be given two or three pints of strong broth, gruel, milk and eggs, to which, if need be, some stimulant may be added, three or four times a day. Drugs can be added to the mixture if necessary. Care must be taken to obtain free elimination by the kidneys and intestines. The skin, too, should receive careful attention. Regular baths, shower baths, and douches are helpful, and may be followed by brisk rubbing and massage. Rest in bed in the early stages is advisable, but the patient should be made to take some exercise, either voluntarily or by massage and passive movements. Care must be taken, if there be any injury, to avoid sepsis. Tonics, cod-liver oil, and cardiac stimulants are sometimes indicated. An abundance of fresh air is advisable, as a prophylactic against tuberculosis. The use of opium in fairly large doses (gr. ss. to ij [gm. 0.05 to 0.20] two or three times a day) is often of decided benefit.

CHAPTER IV

PSYCHASTHENIA

(Insanity of Doubt. Reasoning Insanity. Impulsive Insanity. Anxiety Neurosis. Abortive Paranoia)

Ætiology.—Heredity is usually regarded as the most important cause of this condition, but the mere existence of some form of nervous or mental disease in the antecedents is of far less significance than the occurrence of some condition in the parents, prior to the birth of the child, which might injure the germ plasma and thus produce defective offspring. Among these conditions are alcoholism, chronic infectious diseases and certain constitutional diseases, such as gout and diabetes. An attack of acute disease during pregnancy in a mother otherwise healthy may, by enfeebling the child, be of most serious consequence. Exhausting influences in a healthy individual, either from acute disease, mental overwork, prolonged emotional strain or injury, may also favor the development of psychasthenia. A defective training, in which the individual is never taught to resist discomfort or misfortune or to control his feelings, and the example of other psychasthenics, especially in the family, are also of considerable importance. It is not surprising, therefore, that there are more women who are psychasthenic than men. The condition may arise after any debilitating experience and may develop at any period of life, but the tendency is apt to appear in early life.

Symptoms.—The most important symptom in psychasthenia is the existence of some idea or obsession, which enters into consciousness without or even against the volition of the person affected, who, in other respects, is still possessed of good intelligence. This idea cannot be dispelled and prevents the normal current of ideas. The patient recognizes it as abnormal and foreign and opposes it with his entire consciousness, but without success. These ideas are often absurd and foolish, or sometimes natural and comprehensible, but false. They may take the form of speculation and questioning about some metaphysical problem, impossible to be solved, why men are only as large as they are, why they are not as large as houses, why there are not two suns, or all sorts of problems about God and eternity. Sometimes the dominant obsession recurs in association with all manner of things, the patient may dwell on all the anatomical details of bones, muscles, blood vessels and viscera, when looking at any person, or may imagine genital organs attached to all objects. Sometimes there is an obsession in regard to crime, a mother may have a persistent idea that she may kill her child, or a man the obsession of suicide. Usually the obsession is associated with some emotional state. The persistent idea is associated with some fear, and the result is that the victim is in a state of alarm and mental depression.

The psychasthenic person is always apt to be anxious and troubled about trifles and to worry over them to a needless extent. He is also afflicted with a mania for precision and exactness, and is not content with accepting the probable solution of any question. He is always in doubt; if he leaves the house he is not sure whether he has locked the door, but he must go back to make sure; then, when he has done so, he is still uncertain and must go back again and again. His thoughts can never attain the certainty of belief, nor can his acts be executed correctly and perfectly. He is always in doubt, always undecided, always unable to make up his mind lest he should be wrong, and always anxious. The possibilities of a mistake, of wrongdoing, are always predominant. His mania for precision, too, is usually not directed toward important matters, but to trifles. He will become agitated and disturbed and insist upon rehearsing his complaints again because he said something happened on Monday which really happened on Tuesday, and then he thinks that perhaps after all it was really Monday, and he is again in distress because his statement was wrong. He feels that all his thoughts and actions are performed with difficulty, that he can do nothing well, that he has lost his will power, that he cannot remember, that he has no real feeling. He has no confidence in himself, he needs constant reassurance, because he never can be sure of anything himself or be sure of doing anything correctly and properly.

If the decision be in regard to some transcendental problem, the result will be a process of mental rumination, the "mania of the why," a constant searching and "argument about it and about" without ever reaching a conclusion; partly because the question itself may be unsolvable, but more often because the patient's doubt and uncertainty preclude the possibility of solving satisfactorily to himself the addition of two and two. The result is that the patient is in a state of constant anxiety and distress. In many cases the obsession is of such a nature that it gives rise to some form of anxiety or fear—the various "phobias" for which a portentous list of names has been coined. The patient may become disturbed by the fear that he has been made conspicuous or the subject of criticism from some physical peculiarity or some trivial action: he is ashamed of his stoutness, his awkwardness, his height, his complexion or his dress; he is beset with the fear that he may blush if he meets any one (erythrophobia). He conceives the idea that some poison or some germ may get on his hands and thus be conveyed to his mouth and contaminate him (mysophobia), hence he must perform endless ablutions and take all manner of precautions to avoid contamination. He has the obsession that dust gets in his throat, and he fears all manner of harm therefrom, and constantly clears his throat and spits to prevent it; then he fears that so much spitting will weaken him and that the fear he has in regard to the dust will weaken his mind. Sometimes it is difficult to trace any special obsession that acts as the origin of the fear. Many patients are naturally timid and fear almost anything that has any element of danger to it (pantophobia). Others develop exaggerated fears of some special object or condition. One person is seized with terror at the idea of crossing an open place (agoraphobia), and is incapable of doing so alone, but can do so readily with a companion. Another one has a similar fear of a confined place (claustrophobia), others in regard to high places (acrophobia). One person cannot behold a cat (ailurophobia), even if it be imitation; another has rose cold at the sight of a paper rose. Occasionally it is possible to trace the origin of this fear to some previous event in the patient's life—for example, a persistent fear of going out, which had confined a man to the house for many months, was explained by the fact that he once fell unconscious on the street.

As a result of these various fears and doubts the psychasthenic may resort to certain movements or actions which become habitual and sometimes persist after the obsession has disappeared. These movements are coördinated and have some definite purpose, but, as a rule, they are exaggerated and actually accomplish nothing. Many of the forms of tic are merely the motor response to some previous obsession. They may be of a defensive nature, like the washing of the mysophobic or the clearing of the throat and spitting of the patient beset with the fear of dust. Certain of the forms of facial spasm are of this character, a spasmodic effort to dislodge something from the face or eye. In other cases the spasmodic movement is for the purpose of solving a doubt; the patient shakes his head to be sure it has not fallen off, or touches the wall to prevent himself from staggering. In other cases the action is symbolic of something—an expiation or a pledge against the obsession of evil action. Since the patient is not sure that he can do anything correctly he may insist that every action performed in his presence be repeated a certain number of times. The performance of the special act seems to give a temporary relief to the obsessed mind, and its prevention is often attended with signs of marked distress. The obsession may also tend to the performance of certain actions, which are often spoken of as irresistible impulses. The impulses toward murder and suicide have already been referred to. Although the patient speaks of these impulses as irresistible and fears that they really are, they usually are resisted, at least in the extreme forms. In the lesser forms, the impulse to steal (kleptomania), to set fires (pyromania), or to indulge in alcohol (dipsomania), which sometimes recur periodically, the patient's power of resistance seems less strong, and he often yields. The psychasthenic may be mentally well-endowed; the memory, the quickness of comprehension, the judgment, the attention, and the capacity for logical thought may all be normal, except so far as they may be affected by his obsessions. During a crisis of mental agitation and anxiety he can, of course, think of nothing else; but if, by any chance, the anxieties and fears are relieved, he shows no mental deterioration, and many of the patients can do considerable mental work.

The obsession is usually quite distinct from the delusion, and the association of the obsession with the outer world is seldom real. A patient may imagine anatomical details whenever he looks at anyone, but he does not actually see them, and he is perfectly aware that he does not—he merely imagines them. He knows that his obsession is unreasonable; it comes into his mind against his will and he struggles against it. The deluded patient, on the contrary, believes his delusion to be perfectly true and reasonable; he accepts it as such and modifies his conduct accordingly. Nevertheless, in some cases the psychasthenic begins to doubt whether he really imagines a thing or whether it may not really exist, until finally actual hallucinations and delusions may develop.

When the mental agitation and anxiety are marked the general condition may suffer and various neurasthenic symptoms may develop. In any crisis of anxiety there are often certain physical symptoms—flushing of the face, rapid pulse and respiration, præcordial distress, tremor, sweating, etc. In addition to these transitory symptoms other and more prominent symptoms are not uncommon—headache, insomnia, loss of appetite, gastrointestinal disturbances, and functional disturbances of the circulation. While the milder cases can perform with some difficulty the ordinary duties of life, the severer cases become unfit for any occupation, and conditions of marked exhaustion may develop. The condition may show decided fluctuations, but is apt to be permanent.

Pathogenesis.—The fundamental condition which leads to the psychasthenic state is the consciousness of inadequacy and deficiency, which may arise from either congenital or acquired weakness. In some of the latter cases, when the condition of weakness disappears, the psychasthenic symptoms disappear with it. Most persons have some tendency to doubt, indecision, and morbid fears, and this tendency is increased by fatigue or illness.

The psychasthenic is conscious that he is physically or mentally enfeebled; he has no confidence in his physical strength to protect him from danger, or in his mental strength to understand correctly and to come to a prompt and definite decision. Janet, who has grouped all these varied mental perturbations under the heading of psychasthenia, considers that the essential trouble is the absence of decision and of voluntary resolution, the absence of belief and of attention, and the incapacity of experiencing any feeling in exact relation with the existing situation; the patient has lost the faculty of appreciating reality by perception or action. There is a lowering of the psychological tension in which certain mental phenomena of a higher order are diminished and certain inferior phenomena are exaggerated. The function of the real, voluntary action with a feeling of liberty and personality, the perception of reality, belief, certainty, the enjoyment of the present moment become difficult or impossible; the subject feels the deficiency and his inadequacy. The inferior phenomena, under the form of doubt, agitations, anguish, and obsessing ideas, become exaggerated and dominate the mental field. Psychasthenia is not, strictly speaking, a disease—it is rather a state of mind.

Prognosis.—The prognosis differs between primary psychasthenia and psychasthenia which develops secondarily to some exhausting or depressing state. In primary psychasthenia the patient is born psychasthenic, and the characteristics of the condition become manifest very early in life. If the symptoms be mild, something can be done to relieve them by proper education, and efforts made to improve the general physical condition will also be of benefit. If the condition be the result of some other condition, it may disappear when the other condition is relieved. The dangers which may result from yielding to morbid impulses have already been referred to. In a few instances, as has been said, the psychasthenic condition may develop into an actual delusional state, but this is rare.

Diagnosis.—The milder cases of psychasthenia have often been classed as neurasthenia, and much of the chapter on neurasthenia in the present work has to do with psychasthenic conditions. There is, however, a distinction which should always be kept in mind, although it is not always possible to decide absolutely whether a given case is to be classed as neurasthenic or psychasthenic. Neurasthenia is a condition of pathological fatigue, leading to an irritable weakness of the nervous system, often characterized by certain definite symptoms—headache, backache, insomnia, circulatory disturbances, gastrointestinal disturbances, sexual disturbances, and incapacity for continuous effort, physical or mental. Not only is the patient easily exhausted by a slight effort, but he is much more susceptible to slight irritations. This condition may bring about a lowering of the psychological tension, consciousness of inadequacy, loss of volition, indecision, anxiety, and morbid fears—a train of psychasthenic symptoms. In the psychasthenic, on the contrary, the pathological fatigue, with the attendant symptoms, does not exist at first, and, if it develop at all, it is only after the long-continued mental depression and anxiety have led to exhaustion. The neurasthenic condition, too, usually develops after some definite cause—long-continued worry, overwork, or previous disease; this psychasthenic state generally begins in early life. The distinction from melancholia is sometimes difficult, but the obsessions usually can be distinguished from the delusions of melancholia or from those of the depressive state of dementia præcox by the fact that the patient recognizes clearly their unreasonableness and shows none of the retardation of thought of melancholia or the mental deterioration of dementia præcox. Only in rare cases will it become necessary to make a diagnosis between psychasthenia and paranoia. The paranoiac, in the early stages of his career, may show various psychasthenic symptoms, but the development of actual delusions and hallucinations will usually enable the distinction to be made. It is sometimes difficult to determine whether the impulsive conditions which lead to criminal acts—kleptomania, pyromania, etc.—are genuine. The discovery of other psychasthenic conditions, the history of the case, and in many instances the purposeless character of the act itself, will enable us to determine the real nature of the condition.

Treatment.—One of the first requisites in the treatment of psychasthenic cases is to put the patient into the best possible condition by rest, forced feeding, massage, gymnastics, and an open-air life. A rest cure, as prescribed by Weir Mitchell, is sometimes advisable. In addition to that a judicious system of psycho-therapeutics is often helpful. The patient should be carefully instructed as to the nature of his obsessions, should be constantly encouraged and taught to disregard them, and should be trained to actions requiring prompt decision. Gymnastic movements to music, requiring action in time with the musical beat, are often helpful. Persistent drill in self-control, in enduring discomfort, in resisting the inclination to impulsive movements, and in overcoming morbid fears, is also helpful. The task of reëducating the psychasthenic, however, requires very much time and patience, and is often fruitless.

CHAPTER V

PARANOIA

(Chronic Delusional Insanity)

Ætiology.—We know little as to the causation of this condition. It is hardly to be regarded as a disease, but rather as an intellectual anomaly, so that it is in all probability of antenatal origin, and the psychopathic heredity is, as usual, called upon to explain its occurrence. Men are somewhat more frequently affected than women, and it is said to develop most frequently between the ages of twenty-five and fifty. Indications of the intellectual anomaly, however, can usually be detected in early life, although the full development may not take place until later. In some cases, however, the full development takes place during adolescence ("original" paranoia). As this anomaly is apt to develop in a special type of individuals, often classed as degenerate, it may be well first to describe the paranoid, or "insane," diathesis before giving an account of the fully developed condition, with the necessary proviso that not all of those who manifest paranoid tendencies develop paranoia.

PARANOID DIATHESIS.—From early childhood the subjects of the paranoid diathesis, who are sometimes classed as "higher degenerates," show certain peculiarities which may lead their associates to regard them as "queer" or "cranky." Their egotism is abnormally developed, they are proud, selfish, overbearing, and conceited. They are apt to be distrustful and defiant, perhaps morose and misanthropic. Their temper is seldom under control, and they readily become passionate and vindictive. They have an exaggerated sense of their own importance and of their physical and mental endowments and abilities, which they feel are not fully appreciated by others. They often are actually rather brilliant, achieve fair success in school or college, exhibit various intellectual capacities, and perhaps attain a fair rank in one of the learned professions. Some of them, indeed, may fairly be ranked among the geniuses of the second or third order, although their paranoid admirers seek to place them in the first rank. Tasso, William Blake, Rousseau, Victor Hugo, and Tolstoi are examples of this type. A careful examination of the intellectual performances of these subjects of the paranoid diathesis will usually show that their ideas are extravagant, visionary, and unreasonable. They take up all sorts of schemes, become disciples of all sorts of "movements," embrace all sorts of new and strange religious faiths. They are enthusiastic about some vague scheme of doing away with all the evils of the world and are filled with optimistic assertions about what should be done, without proposing any practical way of doing it; and when their schemes meet with criticism they become excited, and urge them all the more, paying no attention to the rational objections advanced. They invent all manner of wonderful machines that will not work. Any irrational propaganda is likely to allure them, especially if it be visionary and transcendental. They are to be found among the disciples of Mormonism, Spiritualism, Christian Science, and "New Thought"; they are ardent advocates of antivivisection, antivaccination, or anything else that is "anti"; they believe in phrenology, astrology, perpetual motion, the squaring of the circle, the Baconian theory, and panaceas for all earthly ills. They lack the power, however, of observing facts accurately and weighing them judicially, and of coming to a sound and rational conclusion after a due consideration of the evidence upon both sides of a question. They fail to have an adequate conception of the truth. There is often a substratum of value in their ideas, their inventions, and their schemes for the welfare of humanity, and some of their ideas may ultimately be adopted, but their plans are usually vague and general, and cannot be made to work without radical alteration. In such a case they ignore the alteration and believe that they have been robbed of the glory of some great idea or invention. They are unwilling, too, to subordinate themselves to authority or to work in harmony with others; they insist upon carrying out their own notions and zealously maintain that their way is the best. They are eager for notoriety and anxious to be regarded as the chief persons in their circle. When they take part in any movement they are unwilling to subordinate themselves to the welfare of the reform they urge so strongly, and will endanger success rather than leave the center of the stage. In all their mental activities can be noted something eccentric, egotistical, and impractical. In only a few cases are their achievements at all of a higher order; in the majority of instances, of course, they are below the average.

Sexual Perversion.—Among the various anomalies presented by the degenerates are those of the sexual sphere, which demand a brief note. Such persons may show an excessive or a diminished sexual desire, or even a total absence of desire. In rare instances there is a true perversion of the sexual impulse, with a total absence of desire for persons of the opposite sex and a definite sexual feeling toward persons of the same sex (homosexuality). In such cases the patients may show a tendency to imitate in dress and manner those of the opposite sex, possibly with the aim of becoming more attractive to persons of their own sex. In addition to this there are various forms of perversion where gratification can be obtained only by the performance of some peculiar or revolting act—by exhibiting themselves (exhibitionists), by indulging with animals (zoöphilists), or with dead bodies (necrophilists), by inflicting cruelty (Sadists), or by suffering cruel treatment themselves (masochists), or by fondling some special object, like a woman's shoe (fetichists). These perversions may be congenital, or they may be merely symptoms of some more pronounced form of mental disease. They are much more common, however, among those who have exhausted the normal enjoyments, or who are deprived of an opportunity for normal indulgence. A diagnosis of actual perversion, therefore, should never be made without very careful inquiry and the discovery of other indications of mental instability.

Symptoms.—The development of actual paranoia upon such a soil as has just been described often begins with vague complaints of somatic symptoms—headache, tinnitus, palpitation, fullness or emptiness in the head, incapacity for mental work, digestive disturbances, and the like. The patient becomes introspective and hypochondriacal and analyzes his symptoms in detail. He notes some alteration in his mental powers, his thoughts do not work as they used, he can no longer direct them. He grows more dissatisfied with his lot, feels that his associates do not appreciate him or care for him as they ought, and that they do not understand his peculiarities. He begins to feel at odds with the people about him; they seem strange, and his relation to them is unnatural or hostile. He becomes reserved and solitary, reads and studies much on subjects of a religious or metaphysical nature often beyond his comprehension. He develops yearnings for something grand, elevated, and noble, an exalted destiny; he dreams of heroic action, of fulfilling some high mission for the redemption of the world, of practicing some new doctrine, of making some wonderful discovery, and the like. His distrust and suspicion of his associates increase steadily, until finally, perhaps after a gradual development of years, he begins to manifest various delusions and hallucinations.

These delusions are most commonly of a persecutory character. Not only do the people about him fail to appreciate him, but persons are actually endeavoring to do him harm, to inflict some physical injury upon him, to thwart his plans for the welfare of humanity, to prevent him from reaching the exalted station to which he is entitled. At first these persons may be indefinite, but later they are recognized as definite individuals or the agents of some organization. The police have received instructions to watch him, to prevent him from going about, to tell tales concerning him so that he will be forced to leave his home. The Jesuits, the Black Hand, the Nihilists have conceived some plot against him. They influence him in various ways, they poison his food, they introduce poisonous gases into his room through holes in the wall, they hypnotize him, influence him by telepathy, or bring electrical influences to bear by wires hidden in the walls, by Röntgen rays, or by wireless telegraphy. They enter his room at night and inflict most terrible tortures upon him, commit the most perverted sexual acts upon him, deprive him of his physical strength, read his thoughts, and strive to make him insane. The delusion may start with some slight basis of fact, but the patient amplifies it and elaborates it into a fantastic series of delusions. The publisher of some trivial pamphlet, which never met with enough of a sale to pay expenses, may be regarded by the author first as having failed to pay what he owed, then to have swindled him out of thousands of dollars, then to have conspired to send him to an asylum, and finally to have joined forces with the Czar of Russia or the Pope to keep him immured and prevent him from appealing to the world for justice. The paranoiac writes countless letters to the authorities demanding justice; he tries to interview the chief of police, the mayor, the governor, the President, or the King. If he is prevented he seeks to execute justice himself; he must call attention to his persecutions and take vengeance on his persecutors; and thus he lies in wait until a favorable opportunity comes, and calls the attention of society to his wrongs by an attempt at assassination.

In another form the delusions may take on a religious character. The delusions of former days, in which witchcraft, demoniacal possession, werewolves, diabolism, Antichrist, and the like were familiar elements, have, however, largely disappeared, and electricity in its modern applications, bacteria, and poisons have taken their place. The patient develops various mystical and transcendental ideas of a religious character, or becomes the disciple of some new and incomprehensible religious cult. He is not appreciated, people ridicule him because of his beliefs, and supernatural influences are brought to bear upon him in various ways. They may be certain forms of temptations to make him renounce his faith or to lead him to sin; these temptations may take the form of sexual allurements like those of St. Anthony, or they may urge him to blaspheme, to commit the sin against the Holy Ghost, or to adore Satan. The delusions take on a persecutory type, but the religious and mystical element is always pronounced. Unlike the victim of delusional melancholia of a religious type, the paranoiac has never committed sin himself; he may be threatened by devils and the flames of hell, but he is too exalted for them really to have power over him, and he is free from the danger of damnation. His exalted egotism is superior to the powers of darkness.

In a third type the delusions have an erotic character. The patient believes himself to be beloved by some woman, whom perhaps he has never met, usually some woman of prominence in the society columns of the newspaper, an heiress, a noted actress, or some one of noble or royal birth. His attitude to her is of romantic, chivalrous devotion; the sexual element has little or no part in it. He worships her from afar and believes that she reciprocates his devotion, but is prevented from manifesting it publicly; she can respond only by mysterious signs, which she and he only can interpret. If she be an actress and play Juliet, it is her method of revealing to him her undying affection. The worship from afar, however, finally proves inadequate; he must write to her and speak to her to make their affection manifest. He may insist upon intruding into her presence, and when prevented it is because his and her enemies wish to keep them apart. Then may follow a series of delusions of a persecutory character, based upon the erotomania.

The further development of their delusions sooner or later is influenced

by their persecutions and their egotism, and leads to a true megalomania, wi
a transformation of the personality. The reason why they are persecuted
because they are of so great importance. It may be difficult to reconcile th
importance with the actual life of the patient himself, and therefore he e
plains it by a change of personality. He is not what he seems to be, but
really a person of great distinction, the descendant of some king or emper
the rightful heir to the throne, or else he has been appointed by God to u
dertake some great mission. His enemies know who he really is, and to wh
he has been called. Satan knows he has been divinely appointed to destr
his power upon earth, and that is why he seeks to destroy him and preve
the accomplishment of his plans.

In all four classes of delusion—the persecutory, the religious, the erot
or the grandiose—there is one common characteristic. The delusions are fix
and thoroughly systematized. They are combined in a permanent system, w
ordered, elaborately reasoned; the consciousness and mental clearness are pe
fectly preserved, the lines of thought are completely ordered, and such patien
can argue in defense of the beliefs most cogently and coherently, with scarce
a flaw in the processes of their reasoning. Their arguments seldom lack
certain plausibility; they can weave various facts into the net of their del
sion so that it seems almost possible. One paranoiac, who for years distribut
a pamphlet about the streets, had most ingeniously attributed all the une
plained events and sudden deaths among public men for a score of years
a mysterious influence plotted by the Czar of Russia. They are all positi
and dogmatic in their arguments, their faith in their delusions is invincib
by any arguments or demonstrations, they are fond of symbols, and interpr
the simplest movements or events as manifestations of secret influences, th
are fond of strange or new words, they write interminable essays in advoca
of their beliefs, and often emphasize them by peculiarities of spacing, a
rangements, italics, letters of varying size, symbolic diagrams, or drawin
of some artistic skill. Their powers of attention, mental application, an
memory are often surprisingly good. The memory, however, is often di
turbed by their delusions and by their propensity to distort everything to
them. They sometimes perform a surprising amount of intellectual wor
especially in the form of writing. Restif de la Bretonne published over for
separate works in two hundred and forty volumes, which he wished print
in a new alphabet with reformed spelling and in types of various sizes. Ha
lucinations occasionally occur, but they are not very frequent and are oft
absent altogether. The most common form are hallucinations of hearing; ne
hallucinations of smell and taste; hallucinations of sight are rather rare. Ill
sions are much more common. The patient interprets everything that he se
or hears as having some reference to his delusion, as a manifestation of h
persecution, or as a symbol of his real greatness. The disposition is depende
upon the delusions; as a rule, paranoiacs are discontented, disturbed, and su
picious, and they may be markedly depressed.

In other forms of paranoia the delusions are less evident. In some case
to which Lombroso has given the name mattoid, the patient has certain idea
of a pseudo-scientific, humanitarian, utopian, or metaphysical character, whic
he holds with the same tenacity that others do their delusions of persecution
with a sublime independence of common sense, and an absolute devotion t

them. Such patients are sometimes able to maintain tolerable relations with the world about them, and perhaps to practice some profession. They are greatly addicted to writing, and their writings generally show an absurdity of argument, a transcendental character, mysticism, symbolism, and a general lack of practical sense. They are fond of dwelling upon obscure relations between things, upon the inner significance of simple events, of using strange words and an involved style, and of advancing platitudes as new discoveries. In them, too, can be found the exalted egotism and the unreasonableness of all paranoiacs, and a tendency to feel themselves unappreciated, if not actually persecuted.

In the litigious form of paranoia, also, the delusions are not marked. The patient shows the same exaggerated egotism and unreasonableness, but his ideas are directed toward litigation. In many cases there has been some legal controversy in which he believes that he has not got his deserts or in which a decision has actually been rendered against him, and there may really have been some injustice done him. This becomes magnified in his eyes, he turns against his lawyer, and employs one lawyer after another in the hope of getting the decision reversed. He complains bitterly of the injustice done him, and accuses the court of prejudice, if not of actual corruption; he argues that his lawyers were incompetent, that they have failed to carry out the proper forms of procedure, that they have been bribed and have swindled him. He is incapable of realizing that at times personal interest must yield to the general welfare. He is wholly unreasonable when anyone tries to prove to him that his complaints are unfounded and that his case has no standing in law. He may haunt the courts for years, trying to get his case reopened, urging lawyers to make new motions, bringing actions against his former lawyers for dishonest practices, and the like. Such patients have much intelligence, their complaints are plausible; they have sometimes suffered injustice, and they have acquired such familiarity with the law and with legal procedure that they can talk on legal matters as well as any lawyer. It often happens, therefore, that their mental condition is unrecognized, and they are allowed to encumber the courts and waste their patrimony without hindrance. A careful study, however, will disclose the egotism, the unreasonableness, the monotony of ideas and the tendency to believe that they are not appreciated or are persecuted, which they have in common with other paranoiacs.

The conduct of the paranoiac is the natural and direct consequence of his delusions. If he believes that his food is poisoned he will refuse to eat; if gases are forced into his room he will stuff the keyholes and cracks with rags and paper; he may wear some device to keep off evil influences, or put cotton in his ears to prevent him from hearing voices. Many actions are performed as symbolic of his ideas. With grandiose delusions his expression and bearing will be in keeping with his exalted rank; he will demand due reverence, he will call attention to his rank by his dress, by ornaments and badges. Many actions are performed as a defense against persecution. At last, he may attack some one whom he believes responsible for his persecution or commit some act to prove to the world his power and his mission.

Other symptoms are insignificant. Except in the cases where the patient refuses to eat, thus impairing his nutrition, the paranoiac may be in perfect physical health. He may show some intolerance to alcohol and other stimulants, or he may show some physical anomalies, the stigmata of degeneracy; but he is often strong and robust, and may live to an advanced age.

We cannot speak of paranoia as pursuing a definite course like an ordinary disease, but there is often a gradual evolution of the mental state which has been divided into several stages. This must be regarded rather as the education of the individual along a certain definite path, the gradual evolution of one series of ideas out of another in a natural sequence. There is apt to be at first a period of subjective analysis, often somewhat hypochondriacal in character, leading finally to a fully developed delusional state. These delusions develop gradually and become more and more systematized. Many of them arise from efforts to explain the morbid condition. At first they are vague—"they" are persecuting the patient; later, the ideas become more definite, and the persecution is due to some man or some organization. At first the tendency is to escape from the persecution, then to defend himself, and finally to attack the persecutor. Later, there may develop grandiose delusions, perhaps as an explanation of the persecutions, with a change in the personality. As time goes on there is more or less mental deterioration, sometimes amounting to pronounced dementia. The progress is usually very slow.

Pathology.—There is no known pathology of paranoia. An attempt was formerly made to trace a relation between paranoia and anomalies in the cerebral convolutions, but the hypothesis found few supporters. The changes in the brain found in old cases can be attributed to other causes.

Prognosis.—The prognosis is hopeless. In the milder cases something may be done by suitable restraint and careful training to regulate the conduct, but the underlying condition remains and usually will affect the processes of thought and render the patient unfit for society.

Diagnosis.—In some cases the paranoiac will conceal his delusions and even remain absolutely mute. It thus becomes very difficult to determine his mental state, and we are forced to rely upon the history as obtained from others, the examination of letters and other writings, if they can be obtained, and a study of his actions. At times sudden movements of the head as if listening, movements of the lips as if replying to voices, actions of defense, an expression of suspicion or fear, may give some hint. If the patient talk of his delusions the diagnosis is usually not difficult. The history of mental peculiarity and of the gradual evolution of systematized delusions without any exciting cause will serve to differentiate true paranoia from that form of confusional insanity sometimes called acute paranoia. In this the hallucinations are more pronounced, visual hallucinations are common, there is often some confusion, and the patient seldom can reason so logically as to his delusions. There is also frequently some exciting cause—alcohol or infection. The presence of catatonic symptoms will aid in distinguishing the paranoid form of dementia præcox. It is still a question whether a very chronic paranoid state ending in dementia is to be regarded as the paranoid form of dementia
x or true paranoia. If the dementia develop rapidly, however, it may be
ed as dementia præcox. Delusional melancholia may have some re-
ace to paranoia. There is, however, a period of depression which pre-
he development of the delusions, and the melancholic patient shows
f the egotism or expansive ideas of the paranoiac, but is depressed,
e, and guilty.

Treatment.—In some of the milder cases, as has been said, the patient can be somewhat influenced by careful training. In the more pronounced cases treatment is of no avail. The paranoiac is one of the most dangerous persons to be at large, especially when he is disposed to attack his persecutors or when he attributes his persecution to some particular individual. The only method available for the protection of society is by permanent restraint in a suitable institution. There, from their mental superiority to the majority of the patients and from their ability to make plausible arguments, they often make trouble and are often able to make successful pleas for discharge to officials unfamiliar with paranoia.

CHAPTER VI

SENILE DEMENTIA

(*Presbyophrenia*)

Ætiology.—The underlying causes of senile dementia are the same as those incident to old age itself, and especially those of arteriosclerosis. The various factors mentioned under the latter heading (*vide supra*) may therefore be recalled here—heredity, hard physical labor, toxins, etc. Although in most cases of senility we can discover some indications of increasing mental weakness, we know very little of the special causes which tend to the development of actual dementia in certain aged persons. In the prematurely old symptoms of senile dementia may sometimes appear at a comparatively early age (presenile dementia).

Symptoms.—The fundamental symptom of senile dementia, as the name implies, is a gradual and progressive failure of the mental faculties leading to marked dementia. The onset is usually very gradual. The patient has begun to show for some time not only the physical symptoms of old age—the thinning and grayness of the hair, the faded, wrinkled skin, the *arcus senilis*, the rigid, tortuous arteries, the deafness, the bent form, the tremor, and the slow and tottering gait—but he has shown the mental characteristics as well. He has begun to lose the power of adapting himself to new conditions, to take new views of the world, to accept new ideas, and to follow out new lines of thought or industry. He is content to remain in the old ruts, and although he can still do good work along the old lines, he is easily fatigued and the creative faculty is lost. His memory for recent events begins to fail, he has difficulty in recalling the names of recent acquaintances, his critical faculty, his judgment, and his powers of decision begin to decline. He is more or less depressed, he becomes apprehensive and hypochondriacal, and fears the onset of disease. His prejudices grow stronger, he is more firmly fixed in his opinions and more obstinate when suggestions are made to him; he also grows suspicious and distrustful of those about him. He does not comprehend quite so quickly, wanders more and more from the subject at hand, forgets readily, and repeats himself frequently. He grows careless in dress, less considerate of others, more selfish in the gratification of his own whims, and querulous at any contradiction.

With the development of senile dementia these conditions gradually increase. The failure of memory grows pronounced, and is the striking symptom of the affection. The patient becomes unable to find his way through familiar streets, or to recall the names of his own family; he becomes markedly disoriented, cannot tell the day or the month, and fails to recognize his own room, insisting that he is in a strange place and begging to go home, asking for his family who have just visited him, and declaring that they have not been to see him for weeks. The attention is markedly impaired and all the mental processes are slow. There is often much confusion. Delusions and hallucinations are not uncommon. The delusions are often of a persecutory character—that his relatives are trying to rob him or to poison him to get his money. He grows more penurious, has ideas of impending poverty, and practices the most niggardly parsimony, depriving his family of the actual necessities of life. While unreasonably suspicious of his family and his old friends, his failing judgment makes him readily gullible, and he becomes a facile prey to swindlers and confidence men. He makes many mistakes in the ordinary routine of his business or invests money foolishly. Although readily allured by strangers, nevertheless he is ready to break with them at the slightest difference of opinion. He is irascible, fault-finding, unreasonable, and impossible to satisfy. Mental depression is a usual symptom. The senile dement is melancholy, hypochondriacal, and readily moved to tears when any sad event is mentioned, or when anything is said that may contrast his present condition with the past. He believes himself to be neglected and appeals to strangers against his own family. At the same time he may have various grandiose ideas concerning himself, his past achievements and his present abilities, and may boast of them in a childish, extravagant fashion. He is apt to dwell upon a certain limited number of ideas and to repeat the same thing over and over again, forgetting what was said a few minutes before.

In some the sexual element plays a prominent part. Delusions of marital infidelity are common, and the dement will tell most improbable tales of astounding and revolting practices on the part of the suspected husband or wife. Sexual desire is sometimes increased; the patient makes suggestive or indecent remarks to casual acquaintances of the opposite sex, or takes improper liberties. Old men will sometimes expose themselves before young girls, or even make indecent assaults upon them. They often make matrimonial offers to casual acquaintances of wholly unsuitable social position, and, if they have money, are sometimes beguiled into marrying them.

There is often a marked deterioration in the conduct. Not only does the senile dement become self-absorbed and care only for his own personal needs and comforts, to the disregard of all the other members of the family toward whom he was once considerate and affectionate, but he becomes neglectful of the ordinary courtesies of life and even of his own personal cleanliness and decency. He will not take the trouble to bathe or to change his clothing, his clothing becomes soiled with the food which he drops at the table, with saliva, or with urine. Some dribbling of urine is a common symptom. He also fails to dress himself carefully, leaves off various articles of clothing, and goes about with his clothing unbuttoned.

Many patients are inclined to be dull, depressed, and apathetic, disinclined to make any effort, and content to stay quietly much of the time in an easy chair or in bed. They pay little attention to what is going on about them, have little to say, although sad and lachrymose if addressed, and complain little of their condition. It not infrequently happens, however, that such patients, who have been very quiet through the day, are noisy and restless at night, getting out of bed, wandering about the house in the darkness, and making loud outcries and complaints. In other cases, the patients are constantly restless and uneasy, they talk garrulously, complain very much of ill treatment, make loud outcries by day, have fits of anger in which they become abusive and profane, and sometimes destructive, and even assault their attendants.

General symptoms are very common. Many senile dements sleep very little, and, as has just been said, are apt to be noisy and restless at night. Headache and vertigo are very common. Apoplectiform seizures are comparatively frequent. These are usually slight in degree and of short duration, but sometimes there may be severe or even fatal apoplectic attacks. Various focal symptoms, hemiplegia or monoplegia, aphasic disturbances, hemianopsia, etc., may follow the apoplectic attack, but these symptoms are often transitory. Convulsive attacks or temporary lapses of consciousness may also occur. The pupils are small and often react sluggishly to light. The reflexes may show some alteration, due probably to coexisting changes in the spinal cord. Pulmonary, cardiovascular, and renal symptoms, such as have been described under arteriosclerosis, are, of course, to be expected.

The progress of the disease is usually gradual, but death may occur at any time as the result of an apoplectic attack. The senile dement is also predisposed to attacks of intercurrent disease which readily prove fatal. If he escape these, the dementia progresses steadily but slowly, and the end may come either in an attack of delirium, or he may pass into a stuporous state with the usual accompaniment of cystitis, bedsores, and general sepsis.

In addition to true senile dementia, senile patients may, of course, be attacked by other forms of mental disease, especially melancholia of the "involutional" type. In addition to this Kraepelin has described a presenile state of slow onset, in which the patient becomes extremely suspicious, develops persecutory ideas which often have a sexual coloring, and shows a profound impairment of judgment. These cases are apt finally to become demented, and are probably merely one manifestation of senile dementia. Under the name of presbyophrenia Wernicke has described an acute curable condition, the symptoms of which are hardly distinguishable from those of Korsakow's psychosis (*q. v.*), but which are not due to alcohol. Most of these cases recover in a few weeks, but some become demented.

Pathology.—In senile dementia the entire brain may show a considerable diminution in weight, from 100 to 200 gm. below the average. The convolutions show atrophy, are flattened and wider apart, the gray matter of the cortex is thinner, and there may be more or less œdema of the pia. Arteriosclerosis of the cerebral arteries is common, and the brain may show various changes in the distribution of the affected vessels. Microscopically, the cortical brain cells show marked atrophy and degenerative changes, with degeneration in the tangential nerve fibers, which may exist with comparatively little arteriosclerosis, and glia proliferation. Miliary aneurisms, small hemorrhages, and areas of softening may also coexist, with the various changes already described under arteriosclerosis. Lacunæ in the brain substance of larger or smaller size are common, especially about the arterioles.

Diagnosis.—The diagnosis must be based upon the evidence of progressive mental failure in a person who has reached old age. Senile dementia may most readily be confused with melancholia occurring late in life ("involutional melancholia"). The presence of marked failure of the memory and the judgment, the increased irritability and childishness, and the variable and trivial character of the depression will usually make the diagnosis possible.

Treatment.—The aim of treatment should be to support the strength by a suitable diet to meet the symptomatic conditions, and to protect the patient from all excitement and physical injury. Especial attention must be paid to the digestive conditions, the respiration and circulation, and the urinary apparatus, in order to avoid the dangers likely to follow intercurrent disturbances in the different organs. Small amounts of alcohol will often have a beneficial effect upon the nutrition and upon the restlessness and insomnia. Opium in small doses is often of advantage. In conditions of restlessness, moderate doses of the bromids or the warm pack will often have a quieting effect. Further details of treatment may be found in the chapter on arteriosclerosis.

CHAPTER VII

IDIOCY AND IMBECILITY

(*Moral Insanity*)

Ætiology.—Idiocy and imbecility are terms applied to conditions of arrested cerebral development either of congenital origin or occurring in infancy before the higher functions of the brain begin to develop. This arrest of development may be due to a great variety of pathological changes in the brain, and, consequently, to many different ætiological factors. It may be congenital or acquired. The congenital forms are due to causes which have already been mentioned as occurring in the parents and leading to injury of the germ plasma (blastophthoria), such as profound disturbances of metabolism, syphilis, tuberculosis, and alcohol. The fetus may also be affected if the mother, during pregnancy, suffer from profound malnutrition, obstinate vomiting, injury to the abdomen, severe emotional disturbance, etc. The child itself may also be affected, during intra-uterine life, by acute or chronic infectious diseases and toxic and inflammatory processes, which lead to various changes in the brain, such as meningitis, encephalitis, hydrocephalus, porencephaly, microcephaly, or microgyria, lobar or diffuse sclerosis, cortical gliosis, etc. These diseases may also cause changes in the vascular or glandular apparatus (endocarditis, endarteritis, glandular aplasia), which may later affect the brain. Difficult labor may lead to meningeal hemorrhage or injury of the brain itself. Of the various causes of acquired idiocy, occurring in early infancy, we may mention general malnutrition, infection, toxic processes, and mechanical injuries.

Symptoms.—It has long been the custom to divide the mentally defective into three classes, according to the degree of their mental inferiority—the idiots, the imbeciles, and the feeble-minded. The classification is somewhat crude and the boundaries of each class are not clearly defined. The idiot cannot be taught to control his sphincters or, according to some writers, to speak; the imbecile can talk in an infantile manner and can care for his most urgent needs, but never reaches any higher mental development than a child or eight or ten; the feeble-minded can speak normally, but are below the normal in general intelligence and are foolish and credulous. Each class naturally may exhibit many different degrees of mental impairment. Tanzi and others have sought to make a different distinction. They maintain that idiocy is due to acquired cerebral disease, while imbecility is congenital, the ultimate form of hereditary degeneracy, and that each form may show various grades of mental deficiency. The idiot is fundamentally a sick person, outside society; the imbecile is simply a degenerate, against society. This distinction, however, cannot be fully maintained. There are many varieties of mental enfeeblement, due, as has been said, to many different causes, and some of these may be distinguished clinically. It seems better to adhere to the old classification, and to describe the main features of idiocy, imbecility, and feebleness of mind, and then to note the distinctive symptoms of the different clinical types.

Idiocy.—The idiot of the lowest grade is incapable of any volition, he understands nothing, he has no ideas. His feeling of hunger and thirst, even, is defective, if not wholly absent. He cannot recognize food or take it himself, he shows no preference for any food; all that he can do is to make movements of sucking and swallow what is put in his mouth. He breathes and makes inarticulate noises, but he recognizes no one, and cannot understand what is said. He reacts little, if at all, to painful stimuli, nor does he appreciate heat or cold. He can neither stand nor walk nor make any purposive movement of his limbs, but, if not paralyzed, he may show various spasmodic and irregular movements. Auditory and visual stimuli have little effect upon him. He soils himself constantly. The idiot of higher degree may show some vestiges of intelligence. He may notice a bright light, brilliant colors, or moving objects. He hears sounds and may understand a few words. He may reject food that is too salt or too hot. He can take objects awkwardly in his hands, and perhaps feed himself in a gluttonous manner; he can walk in an ungainly fashion and be taught to imitate certain movements. He shows a little memory, recognizes his attendants, and has a little affection for them. There is sometimes a little power of attention, so that he is capable of a small amount of training, but not enough to enable him to prevent soiling himself or to perform any definite task. At times there are violent outbursts in which he seems in a rage and becomes destructive. In a few instances the idiot is able to repeat a few words, parrot-like, but there are usually marked disturbances of articulation, so that the words are scarcely to be understood.

Physically, the idiot usually presents a repulsive appearance. The head is often misshapen, very small, with a low, retreating forehead (microcephaly), or much enlarged, as in hydrocephalus. The eyes are sunken and set near together, there is often nystagmus or strabismus, with a vertical fold of skin at the inner canthus (epicanthus), giving a Mongolian aspect. The pupils may be unequal, the irides diverse in color, or perhaps showing a coloboma. The ears are ill-set and badly shaped. The mouth is half open, the tongue seems too large and projects, the subject is constantly drooling, and the teeth are irregular and carious. The whole face is ugly and expressionless, and often distorted by grimaces. The limbs are ill-shaped and may show various deformities—club feet, syndactylia, polydactylia, etc. Anomalies of the genitals are also common—cryptorchidism, hypospadias, etc. The special senses are often defective, and there may be analgesia or anæsthesia. Paralysis is common, often of a spastic character. In certain forms of idiocy, epileptiform attacks may occur with greater or less frequency. Pulmonary and gastrointestinal disturbances are very readily produced in the idiot, and he is apt to succumb to some form of pulmonary disease, often tubercular, at an early age. In many cases the sexual apparatus is undeveloped; the male idiot is impotent and sterile, the female never or very rarely menstruates, and the uterus is undeveloped; in other cases, unfortunately, the sexual function is active, and the idiot may masturbate freely or procreate his kind.

IMBECILITY.—The imbecile is capable of a limited amount of education: he can be taught to control his sphincters, to dress and feed himself, to walk, talk, and use his hands, to read and write, and to count. Imbeciles are usually backward in early childhood; although they may walk at about the average age, they do not talk until much later, and throughout life may show many defects in articulation, stammering, inability to pronounce certain letters, etc. If sent to school, their inability to keep up with other children soon becomes evident. The memory is usually very poor, although they may show a remarkable memory for a certain class of facts. The attention is defective, and events seem to make only a transitory impression upon them. They can be influenced, for example, by the present threat of punishment, but they lack the vivid memory of former punishments or the conviction that punishment is inevitable, to prevent them from committing the act again. The judgment is exceedingly defective, the reasoning power very slight. They are governed almost wholly by the impressions of the moment and seek to gratify every impulse that arises. The whole mental development is that of a little child. They recognize their comrades and surroundings, and often show a childish affection for those with whom they live, but the affection is neither deep nor lasting, and although they may show some grief at separation, it is easily assuaged. Many of them show a striking lack of sympathy and affection, and are disposed to be cruel to their pets or playmates, the cruelty sometimes taking revolting forms leading to torture, mutilation, and even to death. Their judgments are all superficial, and they are incapable of any abstract ideas or general conceptions. They can be educated only to a slight degree. The writing is extremely defective, the spelling bad, and they can read only simple words, and that very slowly. They seldom are able to count above a hundred or to get beyond very simple addition and subtraction. In their play, they are pleased only with infantile toys, rattles, blocks, and bright-colored balls; they cannot join in sports requiring concerted action or manual dexterity. The same is true in regard to work; they can do simple tasks under supervision, but they can never be trusted to carry out any piece of work by themselves. Many of them have no conception of the truth, but lie even when their falsehood is obvious. They will steal also with a similar disregard of detection or appreciation of the consequences. The feeling of shame or of modesty is also little developed. They exhibit no shame when caught in the commission of some forbidden act, but laugh foolishly; they have little or no modesty in regard to exposing themselves, and often are absolutely without shame in regard to sexual indulgence. Some imbeciles are gentle, quiet, and tractable, and, although they need supervision, give comparatively little trouble. Others, however, are irritable, ill-tempered, restless, and destructive, and seem to delight in malicious mischief. They sometimes are irascible and may become violent and dangerous. Nevertheless, they are usually timid and apprehensive in the presence of superior force. The imbecile shows less of the physical characteristics that have been attributed to the idiot, and he may show none at all. Some imbeciles, indeed, may be physically well-shaped and even attractive. Various anomalies, the so-called stigmata of degeneration, are not uncommon. The sensibility to touch, temperature, and pain, the special senses, the feeling of hunger and thirst, may be somewhat deficient, but are often fully developed. Convulsions and paralysis are less common. The imbecile is often in good physical condition, and may be fairly long-lived.

In this connection a word may be said in regard to a special class of imbeciles, who, although in other respects they present the mental characteristics above described, show somewhat remarkable attainments in one special direction. They are spoken of as *idiots savants.* Some of them show a marvelous memory, an ability to repeat lists of names, dates, or schedules of trains or of the fire department; others are "lightning calculators," who can multiply six figures by six figures or extract the cube root of large numbers correctly in their heads in a few seconds; others still are musical prodigies and can play or sing with remarkable skill; very rarely they show some artistic ability, like Gottfried Mind, the "cats' Raphael," who, although imbecile, was a successful painter of cats. In every institution for idiots there are a certain number of inmates who have some faculty developed considerably above the average of their other mental endowments, without, however, attaining the remarkable ability of the *idiot savant.* Even with the *idiot savant,* his knowledge is sterile; he can play marvelously, like Blind Tom, but he has no critical appreciation of music, cannot compose, and perhaps cannot read a note; he can perform wonderful calculations with lightning-like rapidity, but he has no conception of true mathematics, algebra, or geometry; he remembers a portentous number of useless facts, but he can draw no deductions from them; his mental gifts are merely a psychological curiosity.

FEEBLENESS OF MIND.—The feeble-minded present all degrees of mental enfeeblement, from the imbecile, on the one hand, to the normally stupid person on the other. It is obviously a difficult matter to make any definite boundaries for this class, and sometimes it is by no means easy to determine the status of the individual case. The sensibility and perception are often completely normal, the memory is usually fair and may sometimes be surprisingly good. The feeble-minded are often seemingly alert, active, and quick in their responses, make a good appearance on first acquaintance, are cheerful and attractive, and sometimes fairly skilled in certain handicrafts and superficial accomplishments. They may even have the remarkable attainments of the *idiot savant.* They lack, however, the power of concentrated attention, and there is usually a very striking deficiency in judgment. The mental failure is often noticeable in the later years of school life. They keep up with the average in the lowest classes, but in two or three years they seem to reach the limit of their power of acquiring school knowledge; they may, for example, learn to add and subtract fairly well and perhaps become familiar with the multiplication table, but most of them find long division an impossible barrier. They sometimes cannot acquire very simple geographical knowledge, the location of neighboring towns, and the method of getting to them. It is often hopeless to trust them to perform any definite piece of work; a feeble-minded girl, who has been taught to cook, and who can cook very well under supervision, if left to herself, may use salt instead of sugar, or, after the dish is prepared, may pay no attention to the oven and let it burn to a crisp. Many of them, however, show considerable skill in certain forms of handiwork, and even considerable ingenuity and persistence in carrying out some special plan. There is usually a marked failure of the power to generalize and to comprehend abstract ideas; the imaginative power is also far inferior to the average. Many imbeciles have a certain tendency to fantasy and to invent curious explanations and tales, but they lack ability to form a rational hypothesis to explain any phenomena, or to imagine the consequences of their actions or the necessity of providing for future needs. Their conversation and the range of their ideas show shallowness and puerility; their reasons are those of the child. Like the imbecile, they are vain, credulous, and timorous. They are often affectionate in a superficial way, but they may also be cruel. Ethical conceptions are frequently deficient: they fail to comprehend the truth, they have little regard for the rights of property, and show little or no modesty. Credulous and easily influenced, they are easily led astray. Many feeble-minded young girls readily give themselves to the first comer and become prostitutes, and many feeble-minded boys, incapable of working steadily without supervision, become criminals or tramps. Variations of the emotional mood are not uncommon—periods of mild excitement or depression, sometimes periods of apathy. In some of the more intelligent actual delusions may occur.

The physical conditions may not show any departure from the normal. Some of the feeble-minded are physically well developed and attractive, and may be strong and robust. Others show, to a less degree, the conditions mentioned under imbecility.

Moral Insanity.—Although in the majority of cases the failure to appreciate moral distinctions goes hand in hand with the general intellectual defect, nevertheless there are cases in which the moral defect predominates. In most, if not in all these cases, to which the much-discussed name of "moral insanity" has been given, careful investigation will reveal a considerable intellectual defect; but it is nevertheless true that the intellectual defect is far less evident, and that, to the ordinary observer, the patient may seem of average intelligence. Such patients may even get through the ordinary schools fairly well, and perhaps show some ability in certain directions. On the ethical side, however, they seem wholly lacking in any appreciation of right and wrong, they show no affection toward anyone, are cynically indifferent to the sufferings of others, and are utterly selfish and cruel. They show no contrition for their acts, no appreciation of the consequences, except as they may cause themselves personal suffering, and no persuasion can convince them that their behavior is wrong. Their selfishness and cruelty may lead to the commission of various criminal acts, some of which may show an absolutely cold-blooded indifference to the sufferings of man or beast. They are incapable of leading an orderly life or pursuing any steady occupation. They are irritable and vindictive yet they are sometimes incapable of carrying out any intelligent plans to gratify their revengeful impulses. They yield readily to impulses, are sometimes childish and irrational in their ideas, are unduly susceptible to drugs and alcohol, and their sexual impulses, which are often exaggerated, may be perverted (Sadism). They show various stigmata of degeneracy and usually drift into the criminal class—the "born delinquents" (*"l'uomo delinquente"*) of the Italian criminologists.

Clinical Types of Idiocy

Cretinism.—This is due to loss of function of the thyroid gland, which is caused either by a congenital aplasia of the gland, injuries to the thyroid, or a specific endemic disease of the gland. Endemic goiter prevails especially in the central Alps, also, but less frequently, in the Carpathians, the Pyrenees, the mountain districts of Central and South America, and the Himalayas. Sporadic cretinism may occur in all parts of the world, and Osler has collected many cases in the United States. The affection comes on in infancy, and in its severer forms the condition is unmistakable. The victims are short and squat of figure, often dwarfs, with prominent belly, "blubber lips, *retroussé* nose sunken at the root, wide-open mouth, lolling tongue, small eyes, half closed, with swollen lids, stolid, expressionless face, and muddy, dry skin." They are idiotic and often unable to talk, walk, or stand alone. In the milder cases the lax, waxy skin, the prominent abdomen, and the failure of development may suggest the diagnosis. Closely allied to these conditions is the myxœdematous infantilism described by French authors. The subject preserves the physical and mental characteristics of a child of ten up to the age of thirty or over. The sexual organs are undeveloped, and the secondary sexual characteristics fail to appear. A careful examination will often reveal slight indications of myxœdema—the diminution in size or absence of the thyroid, the thick lips, thick, dry skin, coarse hair, etc.—thus distinguishing it from the angioplastic form of infantilism due to defective development of the arterial system.

Mongolian Idiocy.—This form of idiocy derives its name from the fact that the child resembles the Mongolian type. There is a general arrest of development. The head is small, flattened, asymmetrical, and brachycephalic, the base of the skull shortened, the face flattened, the cheek bones broad and flat, the nose small and flat, the palpebral opening oblique, with a vertical fold of skin at the epicanthus, the cheeks red, the hands short, stumpy, and cyanotic, the tongue enlarged and deeply fissured, the nasal respiration impeded, and other stigmata of degeneracy are common. Congenital heart disease is very common. There are often periodical variations in the mental condition, and the defect is apt to increase rapidly about the school age. They rarely live to adult age.

Epileptic Idiocy.—This form, as the name implies, is attended with frequent convulsions, and may be regarded, perhaps, as an extreme form of epilepsy. It may occur, however, in connection with various lesions in the brain, syphilitic, vascular, etc.

Paralytic Idiocy.—This is merely the mental manifestation of the more profound injury to the brain which sometimes occurs in the cerebral paralysis of children (see page 525), and especially in cerebral diplegia. The latter may sometimes appear as a progressive disease in several members of the family. Choreiform and athetoid movements are common, and convulsions also occur, so that a paralytic idiot may also be epileptic. The mental conditions in these two forms of idiocy vary greatly in degree.

Hydrocephalic Idiocy.—(See the chapter on Hydrocephalus, page 557.)

Infantile Dementia (*Dementia præcocissima*).—Cases have been reported in which, after the child has developed normally to the age of three or four, dementia rapidly developed, which increased to profound idiocy. Excitement, stereotyped attitudes, tic-like movements, and loss of speech are constant symptoms. It may be a form of dementia præcox occurring very early in life.

Amaurotic Family Idiocy.—This form of idiocy, first noted, so far as its ocular manifestations are concerned, by Tay, and later fully described by Sachs, usually occurs in several members of one family, and has thus far been observed only among Hebrews. The children are born healthy and do well up to the age of four to six months. Then they become listless and apathetic and show signs of failure of vision. They soon become paretic or paralyzed in the greater part of the body, the paralysis being either flaccid or spastic. The reflexes may be normal, deficient, or increased. They become totally blind, and the ophthalmoscope shows a cherry-red spot in the region of the macula, with subsequent atrophy of the optic nerve. There is often unusual sensitiveness to sound. The child becomes marantic and dies at the age of two years. Spielmeyer and Vogt have described a juvenile form, beginning between the ages of eight and twelve, and occurring in other than Hebrew families as well. The children become blind, but there are no changes in the macular region. They die in a few years.

Pathology.—The pathology of idiocy and imbecility is exceedingly varied, as has already been said. In some cases few or no changes are to be found in the brain. This is the case with cretins and other forms of idiocy due to affections of the glandular system. The pathology of the Mongolian type of idiocy is obscure, but there may be a general hypoplasia and diminution in the size of the brain, and retarded development of the bones. It has been thought that there was some defective activity of the thyroid gland, but this is uncertain. The pathology of the epileptic and paralytic forms of idiocy is very variable. In the first place, we may find the various changes which give rise to cerebral infantile paralysis, meningeal and intracranial hemorrhages, cyst formations, thrombolic or embolic softening, tumors, and secondary porencephaly. Besides these there may be various forms of atrophic lobar and diffuse sclerosis, affecting certain convolutions, an entire lobe, or even a whole hemisphere. In rare instances the sclerosis may assume a hypertrophic, tuberose form. The brain is dense, often somewhat enlarged, and heavier than normal, and nodules of sclerosis may be felt throughout the brain and may be seen protruding from the surface, which often has a distinctly nodular appearance. These nodules of sclerosis are due to a hyperplasia of neuroglia with abnormalities in the cell formation, and are akin to actual glioma. In these cases tumors are sometimes found in the heart, the kidneys, and the skin, which may enable the diagnosis to be made during life. The nodules occur in the summit of the convolutions and in the walls of the lateral ventricles, while the changes in atrophic sclerosis are more marked at the bottom of the sulci. In both forms of sclerosis the vascular system shows comparatively few changes. In addition to this all forms of malformations of the brain may occur. The anencephalous child seldom lives more than a few hours, but cases occur in which certain parts of the brain are lacking—the corpus callosum, the cerebellum, etc.—and sometimes the subject may live for some time. In many instances the brain is abnormally small (microcephaly), and the cortex may show very few convolutions, or, in rare instances, may be quite smooth. In other cases the brain may be unusually large (macrocephaly). This is due either to hydrocephalus (*q. v.*) or to hypertrophic sclerosis. The changes in amaurotic family idiocy are chiefly microscopic. There are apt to be certain anomalies in the arrangement of the fissures and convolutions, indicative of defective cerebral development, but the chief change is a degeneration of all the ganglion cells throughout the central nervous system, the cortex, the cranial nerve nuclei, the gray matter of the cord, and even the spinal ganglia. There is swelling of the cell bodies, the dendrites, and the hyaloplasm, with destruction of the neurofibrillæ; but the swelling does not affect the axis-cylinder processes. There is in addition a degeneration of fibers in the white matter of the hemispheres and of the pyramidal tracts from the internal capsule to the spinal cord, which Sachs believes to be due to arrested development, the child having a very limited capacity for normal development. The precise nature of the disease is still uncertain. It is not due to syphilis and not of toxic origin. The changes in the juvenile form are similar in character.

Prognosis.—The prognosis of idiocy and imbecility is dependent upon the type of the disease and the severity of the condition. In cretinism and myxœdematous infantilism remarkable results have been obtained by the administration of thyroid preparations. The physical appearance has been completely changed and the patients have made remarkable progress in mental development. If the trouble be of syphilitic origin, distinct benefit sometimes follows antisyphilitic treatment. With these exceptions the probability of cure is very slight, and little can be done in the way of benefit in the severer grades of idiocy, but in the milder grades very much benefit can be expected from proper educational methods. As a rule, the idiot and many of the imbeciles are short-lived, succumbing before thirty to some intercurrent disease.

Diagnosis.—The diagnosis of the more pronounced forms of idiocy and imbecility is usually easy, after the child has reached the age of two or three years. The aspect, the inability to talk, the motor disurbances, and the lack of intelligence are manifest indications of the defective condition, and the diagnosis is often facilitated by malformations of the head, paralysis, convulsions, and stigmata of degeneracy. The rapid mental failure, paresis, and marasmus, and the occurrence in several children of one Hebrew family will often make it possible to diagnosticate amaurotic idiocy without the corroborative proof afforded by the ophthalmoscope. The diagnosis of the higher grades of imbecility and feebleness of mind, and especially of so-called "moral insanity," is more difficult and also far more important. The symptoms often do not attract notice until the child has reached the school age, and perhaps not until toward puberty. A careful study of the child's intellectual powers, often with the aid afforded by an intelligent teacher, will usually reveal the mental defect. In certain cases of "moral insanity" the diagnosis may be possible only after prolonged study, after due consideration of the motiveless acts, the lack of contrition and any ethical feeling, combined usually with more or less mental deficiency. The special features of the different forms of idiocy have been considered above. It is important to note any indications of hereditary syphilis, and, where the question of syphilis arises, to use the Wassermann test.

Treatment.—Cases of sporadic or endemic cretinism and of myxœdematous infantilism should be given the thyroid treatment, as in other forms of myxœdema (*q. v.*). This may have to be continued for years. Under this treatment many cases have been practically cured, but it is important to begin the treatment in early life so that the child may develop normally and obtain an education in the earlier years of life. The thyroid treatment may also be tried in cases of Mongolian idiocy, but the results are less manifest. In cases of syphilitic origin much benefit is sometimes obtained from the use of antisyphilitic remedies. In rare cases of traumatic origin trephining is sometimes indicated. Surgical procedures in microcephalic cases (linear craniectomy) are of no avail. Aside from the myxœdematous and syphilitic cases medicinal treatment can accomplish very little. In many cases, however, very remarkable results may be obtained from suitable educational methods, such as are employed in the best institutions for the feeble-minded. The patients are taught to care for themselves and to work under supervision, so that they become partly or wholly self-supporting. In some instances they can be so developed that they are able to go back into the world and care for themselves. Many of the feeble-minded, however, especially young women, are so apt to be led into evil practices, and many, too, are morally so defective and have such evil tendencies, that it is essential for the welfare of society that they should be permanently under restraint in some suitable institution for the feeble-minded.

APPENDICES

APPENDIX I

PELLAGRA

Pellagra is a noncommunicable disease with digestive, cutaneous, and cerebro-spinal symptoms. Since 1907, medical interest in it has become greatly aroused in the United States, because it has been found to be quite extensive in the South and to appear occasionally elsewhere. It has been reported from thirteen States. The grand total, however, is probably not very large. C. H. Lavinder believes that there are some 10,000 or 12,000 cases in the United States, as compared with an annual total of 350,000 cases of typhoid.

There is a distinct relation between Indian corn or maize and pellagra. The disease was first described in Europe about the time that maize began to be a staple article of diet. It is found in the north of Italy and Spain, in the south of France, and in Roumania. It prevails also in Egypt and exists to some extent in South Africa. Predisposing causes are poverty, worry, bad housing, alcoholism, and the like; but everywhere the essential factor is the habitual ingestion of damaged maize. The peasants of southern Italy are more wretched than those of Lombardy, but they escape pellagra, while in the north, where the season is less favorable to the thorough ripening of the corn, pellagra prevails. In some places, such as Burgundy, where the climate is not very favorable for maize, yet the greatest care is taken to dry it before storing, there is no pellagra, except after a particularly bad harvest (Allbutt). Doubtless there have been many cases of pellagra in the United States for years before Searcy and others called attention to its existence, but it is affirmed that of late our farmers have been less careful than formerly to allow the ears of corn to mature fully before harvesting them. Sambon accuses the sand fly of transmitting the disease.

The exact nature of the poison is unsettled. It is probably a toxin generated in the corn by a fungus. It has been suggested that the toxin develops in the intestines on a diet of spoiled corn. The possibility of an infection has been thought of. Nichols reports the finding of a spore-bearing bacterium which survived steaming for two hours; and Wood speaks of a streptobacillus which resisted a temperature of 90° C. for one hour. Such germs might infect the gastrointestinal tract despite cooking. Protozoa are also suggested.

Domestic animals, except swine, are poisoned by being fed on bad corn. In human beings women are somewhat more often affected than men, and adults in middle life more often than children, although the disease is not rare in those over ten years.

The pathological changes are fatty degeneration and atrophy of the viscera; atrophy and ulceration of the intestines; atrophy, sclerosis, and pigmentation of the skin; degeneration of the spinal cord; and, generally, involvement of the membranes of the brain and cord (Bellamy).

The early symptoms, which appear in the spring, are headache, vertigo, languor, depression, anorexia or bulimia, salivation, stomatitis, and a smooth, red tongue, often with bluish specks. There is dyspepsia, with nausea, vomiting, and intractable diarrhea. Sometimes nurses make the diagnosis from the stools, which are slimy, green, and of a peculiar offensive odor. The temperature is subfebrile. The knee jerks are usually increased and often unequal. In ten or fourteen days, there appears a symmetrical erythema on the parts of the body usually exposed to sunlight (but not absolutely confined to such parts). It is seen on the hands, arms, face, neck, and, if the person goes barefoot, the feet. It is much like sunburn or like a burn of the first degree, but the subjective disturbance is less. There may also be vesicles or bullæ, and the latter may leave indolent ulcers (πέλλα, skin, ἄγρια, malignant). The blood shows no characteristic changes.

Toward the end of summer the disease abates and during the winter the patient may seem perfectly well, but the symptoms recur with the spring. The spinal symptoms include paræsthesiæ, paresis, paralysis of the bladder, a paralytic or spasto-paraplegic gait; the cerebral or mental, vertigo, epileptiform convulsions, confusional insanity, and dementia. The final stage is one of wasting and cachexia, terminated by diarrhea.

There is an acute form, with pain in the epigastrium, headache, nausea, vomiting, severe diarrhea, with bloody stools, fever, delirium, and a typhoid course, terminating fatally in ten days or six weeks.

As to prognosis, the earlier the case is recognized the better. The chronic type without mental involvement has the best outlook. The recovery of patients who have suffered for three or four seasons and have become insane is almost hopeless. The acute typhoid form usually does not recover.

It is highly desirable that the physician should be able to make the diagnosis early, independently of any cutaneous lesions, for these are not the first manifestations of the disease, and, furthermore, some cases are said to occur without them (*pellagra sine pellagra*). The "bald" red tongue, the obstinate diarrhea, and the exaggerated knee jerks, may be emphasized in this connection. There is little doubt, says Lavinder, that pellagra has existed in the southern United States for at least twenty-five or thirty years, and been regarded as an unusual manifestation of tuberculosis, syphilis, malaria, acute delirium, hookworm disease, dermatitis exfoliativa, eczema, and the like. Allbutt mentions the possible consideration of general paralysis, tabes dorsalis, leprosy, and Addison's disease. Doubtless in most cases the disease will be recognized, if it is borne in mind.

Treatment demands, above all, a change to proper hygienic conditions. No maize whatever should be eaten. There is a possibility that, in the sick, good corn may perpetuate or renew the disease. The blood serum of cured cases has been successfully used in the treatment of typhoid pellagra. Cole has reported the transfusion of blood from a cured individual with good results. Fowler's solution and atoxyl have been employed without much benefit. Chlorid of sodium was recommended by Lombroso.

A bibliography is appended.—[V.]

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APPENDIX II

SUMMARY OF THE SYMPTOMS AND TREATMENT IN CASES OF POISONING

1. **Sulphuric Acid.**—Mucous membrane of mouth, throat, œsophagus, and stomach deeply corroded. In the worst cases rapid death, ushered in by convulsions and asphyxia, or, more rarely, from perforation into the mediastinum with suppurative mediastinitis, or from peritonitis consequent upon perforation of the stomach. Usually the case is more protracted. The mouth and throat are whitened, or, in severe cases, blackened. They are soon attacked by an intense ulcerative inflammation. Deglutition is extremely painful, and there are most distressing choking and retching. The vomitus contains black lumps. Profuse salivation. Pain along the œsophagus. Dangerous respiratory disturbances from resulting œdema of the glottis. Abdomen usually distended and very tender on pressure. There may be intestinal discharges of a bloody character resembling dysentery. Urine is generally scanty, and often contains albumen and blood. Fever. Collapse. Small and rapid pulse.

In mild cases, slow recovery, the necrosed tissues gradually sloughing off. Cicatricial stricture of the œsophagus or in the neighborhood of the pylorus frequently ensues and may prove fatal. Neuralgia, hyperæsthesia, and various other nervous disturbances may also occur as sequelæ.

In fatal cases, the autopsy discloses necrosis, ulceration, and inflammation in the upper portion of the digestive tract. The mucous membrane of the stomach is usually coal-black. Well-marked parenchymatous degeneration of the liver and kidneys (nephritis). In later stages, extensive cicatrices.

Treatment: If used at all, the stomach pump must be introduced very cautiously, for fear of causing perforation. The best remedy to give at once is several teaspoonfuls of magnesia in water, or a few drops of dilute liquor sodii hydroxidi in mucilage. Later, the symptoms are to be combated with bits of ice, disinfecting mouth washes and gargles, tonics, and cautious feeding with milk, eggs, etc. If stricture of the œsophagus develops afterwards, an endeavor should be made to dilate it with bougies.

2. **Hydrochloric and Nitric Acids.**—Symptoms similar to those of sulphuric acid. The most prominent symptoms are the local ones, intense stoma-

titis, pharyngitis, etc. There is usually albuminuria, with casts and blood in the urine. Nitric acid stains the corroded spots, especially the angles of the mouth, yellowish; and the vomitus may have the same tinge. In poisoning from fuming nitric acid the inhaled vapors cause affections of the respiratory passages (œdema of the glottis). Prognosis and treatment as in poisoning from sulphuric acid.

3. **Nitrous and Sulphurous Acid Fumes.**—Intense local inflammation of the air passages. Violent dyspnœa, cough, abundant bloody or yellowish expectoration. There may also be grave nervous disturbances and collapse. *Treatment:* Symptomatic; sinapisms, narcotics, expectorants, and inhalations of alkaline sodium-chlorid solution.

4. **Oxalic Acid.**—Local corrosive action similar to that of the other acids, only less severe. In severe cases collapse from cardiac paralysis. Apt to occasion certain nervous symptoms—formication, anæsthesia of the finger tips, tonic and clonic convulsions, trismus, and tetanus; later paresis. There is sometimes sugar in the urine, and nephritis may develop. Anuria is frequent, and is due to plugging of the renal tubules by calcic oxalate crystals. The treatment should be symptomatic, and should also include the administration of preparations of calcium—liquor calcis, calcii carbonas, or even egg shells—to form insoluble calcic oxalate. Magnesia is also useful. Subcutaneous or intravenous injection of salt solution, containing ʒss. to ʒj (gm. 2 to 4) per quart.

5. **Ammonia.**—The fumes affect the air passages chiefly; the solution, the upper part of the digestive tract. The specific local effect is the production of an intense croupous inflammation of the mucous membrane. Accordingly, the symptoms are salivation, dysphagia, vomiting of strongly alkaline matter, and diarrhea, or cough, dyspnœa, etc. The urine does not become alkaline, as the ammonia is converted into urea. In severe cases there is collapse, with rapid pulse, and such nervous symptoms as pain, paræsthesia, vertigo, convulsions, etc. Permanent strictures are rare. *Treatment:* In fresh cases the stomach pump. The cautious use of acids—for instance, acetic or citric. For the rest, symptomatic measures.

6. **Caustic Potash or Soda.**—Symptoms and treatment as in poisoning from ammonia. In contrast to acid poisoning, it is to be noted that alkalies do not withdraw water and precipitate albumen, but dissolve it. The corroded spots are therefore not dry and brittle, but softened ("colliquation"). Urine alkaline. Permanent strictures frequent owing to the deep action.

7. **Potassium Nitrate.**—Vomiting and diarrhea. Severe abdominal pain. Collapse, with cold skin and rapid, thready pulse. Occasionally the pulse is slow. Nervous disturbances, such as painful muscular contractions, and, in severe cases, convulsions and coma. *Treatment:* Symptomatic; opium and other narcotics, stimulants (camphor, ether), and bits of ice. Abundance of fluids. Sodium-chlorid infusions.

8. **Chlorin Gas.**—Violent convulsive cough. Bloody expectoration. Spasm of the glottis. Dyspnœa. Darting pains through the chest. Sneezing and profuse flow of tears. In severe cases pneumonia. *Treatment:* Fresh air. Inhalation of warm aqueous vapor, or of ammonia. Narcotics.

9. **Chlorate of Potassium.**—Lethal poisoning has been noted in children after only gr. lxxv to ℥ss. (gm. 5 to 15). Destruction of the red blood cor-

puscles and conversion of the hemoglobin into methemoglobin. In severe cases, jaundice, cyanosis, dyspnœa, and diarrhea. Nephritis. Methemoglobin demonstrable in the urine and blood. Uræmia. Milder cases may recover. *Treatment:* Gastric lavage. Laxatives. Promotion of diuresis (mineral waters). Cardiac stimulants. Administration of alkalies.

10. **Iodin.**—1. *Acute iodism,* as seen, for instance, after the injection of large amounts of tincture of iodin into ovarian cysts: collapse, with pallor and cyanosis, and small and very rapid pulse. Vomiting. Often marked dyspnœa. Suppression of urine. Sometimes hemoglobinuria. Later, the skin becomes very red. There is albuminuria; also sore throat, coryza, conjunctivitis, severe frontal headache, and cutaneous eruptions. 2. *Chronic iodism,* caused, for example, by long-continued internal administration of iodin or potassium iodid: coryza, conjunctivitis, catarrh of the frontal sinuses, sore throat. Gastric symptoms. Vertigo, headache, and similar nervous phenomena of a mild character. Acne or erythema, sometimes in the form of erythema nodosum. After prolonged use of the iodin preparations, a peculiar condition, due, perhaps, to the action of the iodin on the thyroid gland, occurs: remarkable emaciation and weakness, associated with continued tachycardia. *Treatment:* In acute cases, white of egg and stimulants. Other than this, treatment must be symptomatic. Prophylaxis demands caution in the internal administration of iodin or its compounds. According to Ehrlich, the symptoms of iodism often disappear rapidly after the exhibition of about ʒjss. (gm. 6) of sulpho-nitro-salicylic acid (sulphanilic acid).

11. **Bromin.**—1. *Acute poisoning* from the fumes of bromin excites the same symptoms as does chlorin gas. 2. *Bromism,* resulting from the long-continued use of potassic bromid, causes languor, debility, mental apathy, and impaired intellectual vigor. The reflexes are diminished, particularly the reflex irritability of the soft palate and pharynx. Bad odor to the breath. Anorexia. Diarrhea. Impotence. Almost invariably a characteristic acne, the onset of which may be delayed by giving arsenic at the same time.

12. **Lead.**—(*a*) *Acute lead poisoning* produces severe gastro-enteritis. The best antidote is sulphate of sodium or magnesium; or phosphates, white of egg, and milk. In fresh cases the stomach pump, or emetics and purgatives. Other than this, symptomatic treatment.

(*b*) *Chronic lead poisoning:* Seen in typesetters, typefounders, painters, polishers, potters, and others. Also in food poisoning from cooking utensils or preserving cans containing lead; has also been observed from tin-foil containing lead (snuff, chocolate), water from lead pipes, etc. General symptoms: Lead line on the gums, lead anæmia, and lead cachexia. In the blood very frequently basophilic granular red blood corpuscles. Important groups of symptoms are: 1. *Lead colic:* Violent colicky pains radiating from the umbilicus. Usually constipation, exceptionally diarrhea. Abdomen concave and hard. Frequent vomiting. Hard, slow pulse. Temperature usually normal. Urine sometimes contains a trace of albumen. Duration, one or two weeks. *Treatment:* If severe pain, opium and hot compresses. Atropin may also be tried. For constipation, enemata and gentle laxatives. Warm baths. 2. *Lead paralysis* (*vide* page 249): Paralysis of the laryngeal muscles due to lead poisoning has been reported. Occasionally atrophy of the optic nerve. 3. *Saturnine encephalopathy:* Sudden development of grave cerebral symptoms; convulsions, coma, delirium, great mental uneasiness, and excruciating headache. Saturnine amaurosis. In severe cases, death. Cerebral lesions are very rarely found postmortem. Treatment is symptomatic. Lukewarm baths, with douching, narcotics, and stimulants. Later, potassium iodid. 4. *Lead arthralgia:* Most frequently attacks the knee. Also seen in the joints of the upper extremities. Sometimes associated with painful muscular contractions. Objective lesions are rarely seen. The treatment consists of warm baths and the administration of potassium iodid. It should be remembered that chronic lead poisoning may occasion gout and chronic interstitial nephritis (*q. v.*).

13. **Copper** (*Blue Vitriol, Verdigris*).—*Acute copper poisoning:* Copper taste. Vomiting of greenish matter, colic, tenesmus, and bloody stools. Also nervous symptoms, headache, vertigo, anæsthesia, paralysis, delirium. Collapse. Dyspnœa. *Treatment:* White of egg, milk, wood charcoal. Magnesia is also valuable. Yellow potassium ferrocyanid, milk sugar. *Chronic copper poisoning* is rare. It occasions gastrointestinal disturbances, colic, and a reddish or greenish discoloration of the hair.

14. **Zinc** (*Zinc Sulphate and Chlorid*).—In acute poisoning, symptoms of severe gastro-enteritis, especially vomiting. Also albuminuria. *Treatment:* Albumen, tannin, and alkaline carbonates. Superheated zinc fumes cause the so-called "smelter's fever" (asthma, chills, fever up to 104° F. [40° C.]). Recovery after three to six hours under diaphoresis.

15. **Mercury.**—(*a*) *Acute poisoning from corrosive sublimate:* The mucous membrane of the mouth, throat, œsophagus, stomach, and intestines is deeply corroded. Metallic taste. Vomiting. From the excretion of mercury by the intestine, diarrhea with bloody and mucous stools and painful tenesmus. From the excretion of the poison through the kidneys, nephritis (albuminuria), ischuria or complete anuria. Recently we have seen acute sublimate poisoning, especially in nurses, who had taken the so-called corrosive tablets. A few hours after the ingestion of the poison, albuminuria was demonstrable. Then there ensued a deceptive stage of seeming euphoria, lasting seven to ten days, and, finally, death from uræmia. *Treatment:* Milk, albumen, narcotics. The enormous swelling of the fatty degenerated kidneys suggests the idea of trying in these cases, which are otherwise hopeless, to save the patient by means of operative division of the capsule of the kidneys. (*b*) *Chronic mercurial poisoning:* Seen in the makers of thermometers, scientific instruments, and mirrors, and in gold-platers. Rarely occasioned by the prolonged use of antisyphilitic remedies. The symptoms are anæmia, emaciation, with great muscular weakness, and gastrointestinal disturbance. In the therapeutic use of mercury, besides the rarer mercurial enteritis, mercurial stomatitis is the most important toxic symptom; onset of foul-smelling necrosis of mucous membrane, with ulceration of mucous membrane of cheeks, jaws, gums, etc. *Treatment:* Immediate discontinuance of mercurial medication; hydrogen peroxid as mouth wash and gargle.

Mercurial Tremor.—Tremor is common in chronic mercurial poisoning and is of special interest. It is usually preceded by a stage of pronounced mental irritability ("mercurial erethism"), and often some mental excitement, fright, etc., may be the exciting cause of the tremor. The tremor itself is a pronounced intention tremor—that is, it is usually not present when the body is kept quiet, but it comes on with all movements, like the tremor in multiple sclerosis. The finer the movements the patient tries to make the more marked is the tremor. Mental excitement usually increases the tremor considerably. In severe cases the tremor is so great that the patients cannot leave their beds. In rare cases there is also paralysis.

Treatment: Dietetic and hygienic. Iodid of potassium, hyoscin, and sometimes galvanism, are also useful.

16. **Phosphorus** (*vide* also Vol. I, page 700.—(1) *Acute phosphorus poisoning,* as from matches: Violent epigastric pain, vomiting—the vomitus smells of phosphorus, and it may be phosphorescent in the dark. After these initial symptoms usually comes a period of comparative comfort, lasting two or three days. Then appear grave symptoms: jaundice, severe pain in the abdomen, hepatic enlargement, fever, feeble and rapid pulse, sometimes gastric or intestinal hemorrhage, cutaneous ecchymoses, hematuria, epistaxis, or metrorrhagia. The intellect usually remains clear, except that just before death there may be drowsiness or convulsions. The urine contains albumen, blood, casts, bile-pigment, and rarely leucin and tyrosin. No urea is excreted. Death occurs usually at the end of one or two weeks, but it may be more speedy. Mild cases recover. Nevertheless, a very grave prognosis should be made in every case at the start. Even in apparently mild cases there may be sudden heart failure. *Post-mortem appearances in acute phosphorus poisoning:* Jaundice. Dark blood. Numerous internal ecchymoses—for example, in the serous and mucous membranes and kidneys. Fatty degeneration of most of the internal organs, including the liver, heart, muscles, and kidneys. *Treatment:* In fresh cases, protracted washing out of the stomach with $\frac{1}{10}$ of one-per-cent solution of potassium permanganate. Laxatives. The best emetic is sulphate of copper. The best antidote is old oil of turpentine (30 to 40 drops in mucilage). Fats and milk should be avoided, as phosphorus is soluble in fats. Narcotics and other symptomatic remedies may also be indicated. (2) *Chronic phosphorus poisoning:* Necrosis of the lower jaw, less often of the upper jaw, extending from caries of the teeth. Necrosis of the bone with exuberant growth of osteophytes.

17. **Arsenic** (Arsenious Acid, Schweinfurth Green, Scheele's Green, etc.). —Lately arsenic intoxications have repeatedly occurred through incautious medical use of atoxyl and similar arsenic preparations. (1) *Acute arsenical poisoning:* Symptoms of intense gastro-enteritis, suggesting cholera. Violent vomiting. Rice-water stools. Severe abdominal pain. Other mucous membranes also may show irritative symptoms (conjunctivitis, bronchitis). Nervous symptoms: vertigo, headache, faintness, twitchings. Cardiac weakness. Cyanosis. Collapse. Not infrequently a cutaneous eruption resembling urticaria or eczema. Sometimes albumen and blood are present in the scanty urine. Severe cases are fatal in one or two days. With regard to arsenical paralysis, *vide* page 250. *Treatment:* At first the stomach pump or emetics (sulphate of zinc). The best antidotes are freshly prepared ferric hydrate in water, two to four tablespoonfuls every fifteen to thirty minutes; ferrum oxydatum saccharatum solubile (P. G.), in teaspoonful doses; calcined magnesia; and, best of all, the compound magnesia and ferric hydrate known as "*antidotum arsenici*" (P. G.), of which one or two tablespoonfuls may be given every fifteen to thirty minutes. (2) *Chronic arsenical poisoning:* Ac-

quired in arsenic works and glass factories, or from arsenical fabrics, wall papers, and artificial flowers. Conjunctivitis, chronic gastrointestinal catarrh, eczema, and cutaneous ulcerations. Anæmia and cachexia, falling out of the hair, loss of sleep. Treatment is purely symptomatic, except as regards prophylaxis. Internally, iodid of potassium. In poisoning with arseniureted hydrogen, hemoglobinuria and jaundice occur, with severe nervous symptoms.

18. **Chromium Salts.**—Yellowish-red desquamation. Gastro-enteritis resembling dysentery. Inflammation of the urinary passages and acute nephritis. In the gravest cases cardiac collapse and death. *Treatment:* Calcined magnesia in water. Gastric lavage with one-per-cent solution of sodium sulphate or hyposulphite.

19. **Alcohol.**—(1) *Acute alcoholic poisoning:* Unconsciousness; anæsthesia; pupils either dilated or contracted, usually not reacting to light; pulse small, sometimes slow; skin cold and clammy; vomiting; stertorous respiration. There may be delirium and clonic convulsions instead of coma. Such cases last three or four days, or even less. Death has been repeatedly observed, especially after exposure to cold from loss of body warmth (dilatation of the cutaneous vessels!), when inebriates fall asleep in the open air. *Treatment:* Bathing and douching. Stimulants, caffein subcutaneously.

(2) *Chronic alcoholism.* (*a*) *Physical and mental debility:* Chronic catarrh of the throat, larynx, stomach, and intestines. Alcoholic tremor. Numerous organic diseases, including cirrhosis of the liver, contracted kidney, heart disease, cerebral disease, and neuritis.

(*b*) *Delirium tremens:* Usually a sudden onset, as in connection with some acute disease or after a traumatic injury. Disordered intellect. Great restlessness, hallucination (vermin, strange voices, etc.), excitement, and loss of sleep; duration, four to five days. *Treatment:* Bathing and douching. Injections of strychnin. The use of chloral and other narcotics should be guarded. Paraldehyd in doses of ♏ lxxv to xc (gm. 5 to 6) is worthy of commendation. Physical restraint should be avoided if possible. The patient may often be allowed to go about the room as he likes, if only he be watched. Alcohol should be given if collapse be threatened. As a rule, sudden and complete withdrawal of alcohol does not act injuriously. (See page 666.)

(*c*) Alcoholic polyneuritis and so-called Korsakoff's psychosis, *vide supra,* pages 273 and 663.

20. **Chloroform.**—Unconsciousness. Loss of sensibility and reflexes. Slow pulse. Pupils dilated. Failure of respiration, and finally of the heart. Danger of heart failure, especially in persons with weak hearts. *Treatment:* Artificial respiration. Injections of strychnin. Stimulants. Counterirritation.

21. **Iodoform.**—(Repeatedly seen from the use of iodoform on wounds.) First of all, nervous symptoms: headache, vertigo, sleeplessness. Peculiar psychoses, maniacal attacks, delusions of persecution, refusal of food. In severe cases, convulsions, deep coma. Very small, rapid pulse. *Treatment:* Symptomatic, by stimulants, baths, etc. Alkalies and atropin are recommended, but their action is doubtful.

22. **Carbonic-oxid Gas** (Coal Gas, Illuminating Gas).—At first, vertigo, headache, throbbing in the temples, ringing in the ears, and spots before the eyes, vomiting. The patient gradually becomes unconscious. Skin pale and

cyanotic. Respiration intermittent. Convulsions, later paralysis. Pulse at first accelerated, later intermittent. Subnormal temperature. The urine has been frequently found to contain albumen and sugar. The carbonic oxid may be demonstrated in the blood by means of the spectroscope. Its color is clear cherry-red (CO-hemoglobin). Subsequently paralysis, disturbances of sensation and of speech. *Treatment:* Fresh air, artificial respiration, inhalation of oxygen, venesection with subsequent subcutaneous or intravenous saline infusion.

23. **Sulphureted Hydrogen** (Sewer Gas).—Headache, vomiting, diarrhea. In severe cases, unconsciousness, dyspnœa, cyanosis, convulsions, and death. The blood is thin, fluid, and black (sulpho-hemoglobin). *Treatment:* Artificial respiration, fresh air, and the cautious inhalation of chlorin gas (chlorin water), venesection, and saline infusion.

24. **Bisulphid of Carbon.**—(Workers in rubber factories.) Vomiting. Severe nervous symptoms. Incontinence of urine, atrophic paralyses from polyneuritis, anæsthesia, mental disturbances, especially loss of memory, convulsive conditions, etc. The red blood corpuscles are destroyed; the black blood contains many flakes of pigment. *Treatment:* Symptomatic.

25. **Hydrocyanic Acid** (Potassium Cyanid; Bitter Almonds).—Characteristic odor of bitter almonds. In severe cases death may occur in a few minutes, with dyspnœa and convulsions. If the course be more protracted, convulsive and extremely slow respiration, the expiratory act being much prolonged, the eyeballs protrude, and the pupils are somewhat enlarged and do not react to light. Cardiac weakness, mucous membranes bright red, unconsciousness. Twitching of the muscles. Trismus. Usually respiratory paralysis occurs before heart failure. *Treatment:* Merely symptomatic. Emetics, artificial respiration, cool douches, stimulants. Experimentally, lavage of the stomach with two-per-cent hydrogen peroxid or potassium permanganate. Also subcutaneous use of hydrogen peroxid in two-per-cent to three-per-cent solution, or intravenously ʒijss. to ℥j (10 to 30 c.c.) of an 0.8-per-cent solution of sodium thiosulphate.

26. **Nitrobenzole** (Oil of Mirbane).—Strong odor of bitter almonds. At first, dizziness. The skin soon assumes a bluish hue, rapidly increasing to the deepest cyanosis. Increasing anxiety, sense of suffocation, and gradual loss of consciousness. In severe cases, death, ushered in by convulsions. In milder cases, gradual recovery. *Treatment:* Stomach pump. Artificial respiration. Stimulants. Inhalation of oxygen. In the Leipsic clinic, transfusion has worked admirably in two cases. The symptoms caused by anilin, antifebrin, phenacetin, etc., closely resemble poisoning from nitrobenzol. The nitrites also form methæmoglobin and act by paralyzing the blood vessels.

27. **Carbolic Acid, also Lysol, etc.**—Corrosion of mouth, throat, and stomach. In mild cases, vertigo, headache, and collapse; in severe cases, coma, preceded in rare instances by symptoms of cerebral irritation. Contracted pupils. Vomiting. Pulse slow at first, then rapid. The urine has a dark, olive-green color. Sometimes hemoglobinuria and nephritis. Carbolic acid is very readily absorbed from the rectum, so that great caution should be employed in its use as an injection. *Treatment:* Stomach pump. Slaked lime, calcium saccharate; also large doses of sulphate of sodium. [Alcohol is an antidote. Administer three or four ounces of whisky at once, then wash

out the stomach with water containing sulphate of sodium. In desperate cases, venesection, followed by infusion of normal salt solution, has been successful.—V.]

28. **Atropin** (*Belladonna*).—Dryness of the mouth and throat. Excessive thirst. Dizziness and headache. Peculiar mental disturbances: hallucinations are particularly frequent. Pupils very widely dilated. Cutaneous erythema resembling that of scarlet fever. In severe cases, pulse enormously accelerated, with violent pulsation in the arteries. Convulsions may occur. Nervous symptoms persist for some time. *Treatment:* As the main antidotes, morphin, tannin, and coffee should be tried. Abundance of fluids.

29. **Digitalis.**—Vomiting. Diarrhea. Pulse very slow (forty beats per minute, or less). Dyspnœa. Symptoms of collapse. Cold extremities, muscular tremor. Somnolence. In the worst cases, sopor and death. Even the milder cases are protracted. *Treatment:* Stomach pump. Tannin, complete rest in bed. Alcohol, atropin. In cardiac failure, camphor, strong *café noir*, ether, ammonia. Counterirritation.

30. **Nicotin** (Smoking; Tobacco Juice; Tobacco Enemata).—(1) *Acute:* Pulse small and slow, syncope, sense of oppression, salivation, vomiting. In severe cases, delirium, loss of consciousness, tetanic spasms, pulse and respiration intermittent. (2) *Chronic* (from excessive use of tobacco): Palpitation, irregular action of the heart, paroxysms of asthma and angina pectoris. Tremor, muscular weakness. Loss of sleep. Visual disturbances (amblyopia, scintillating scotoma). Gastric and intestinal disturbances, chronic catarrh of the pharynx and larynx. Continued and excessive smoking probably favors the occurrence of arteriosclerosis. *Treatment:* In acute cases, stimulants. Chronic poisoning necessitates the giving up of tobacco. Further treatment is symptomatic.

31. **Strychnin.**—Violent tetanic reflex convulsions. Exaggeration of the cutaneous and tendon reflexes. Trismus. Opisthotonos. Pulse small and very rapid. The convulsions come in paroxysms, with intervals between them. The intellect is usually perfectly clear. Recovery occurs only in mild cases. *Treatment:* Emetics, stomach pump. Tannin. Tincture of iodin. Castor oil. The convulsions are to be combated by narcotics (morphin, chloroform, chloral).

32. **Coniin** (Hemlock).—In severe cases convulsions, then general paralysis, especially of the respiratory muscles, loss of consciousness, and death. Pupils dilated. In milder cases confusion, muscular weakness, vomiting, and diarrhea. *Treatment:* Emetics, tannin, stimulants, artificial respiration.

33. **Morphin** (Opium).—(1) *Acute:* Begins with fatigue, headache, darkening of the visual field, nausea, vomiting. Then coma comes on with slow, stertorous, sometimes irregular respiration. Muscles completely lax. Pupils usually very small. Pulse often slow, but in other cases rapid and small. Toward the end of life Cheyne-Stokes's respiration. Danger of pneumonia. In milder cases only vomiting, stupor, headache, etc. *Treatment:* Emetic (futile in coma), stomach pump. [One-per-cent solution of permanganate of potassium.—V.] Tannin. *Café noir.* Atropin may be tried as a physiological antidote. Stimulants (camphor, ether) are best, also warm baths and shower baths; artificial respiration. (2) *Chronic* (morphin habit): Emaciation, anæmia, headache, vertigo, wakefulness. Tremor. Psychical disturbances

(mental and moral weakness). Unconquerable longing for morphin; and, if this be suddenly withdrawn, the appearance of grave symptoms. The suspicion of chronic morphinism is often created by the appearance of the skin with its numerous scars, pustules, and furuncles resulting from many self-performed injections. To break up the morphin habit is usually possible only in hospitals and special asylums. The withdrawal of the drug is abrupt according to the practice of some, and gradual according to others. For particulars, consult monographs.

34. **Cocain.**—(1) *Acute:* Psychical excitement and confusion, accelerated heart action and respiration, præcordial anxiety, twitching, contractures, even epileptiform spasms; in other cases severe collapse with anxiety, pallor, and syncope. *Treatment:* Tannin, stomach pump, amyl nitrite; morphin in convulsions, atropin in collapse. (2) *Chronic:* Rapid cachexia, loquacity, mental degeneration, anxiety, hallucinations, especially of tactile nature ("cocain animals"), delusions. *Treatment:* Withdrawal.

35. **Ergot** (Ergotin).—(1) *Acute:* At first nausea, vomiting, colic, and diarrhea. Then vertigo, headache, and muscular weakness. Pulse slow. In severe cases, sopor, disturbance of respiration, and sometimes death. *Treatment:* Emetics and purgatives. Tannin. Ether, camphor, and *café noir* as stimulants. (2) *Chronic ergotism* (crawling sickness): Gastric symptoms, vertigo, languor, cardiac weakness. The nervous disturbances are, however, especially interesting. Of these, paræsthesia (the "crawling") has been recognized longest. Recently, attention has been attracted to the fact that the nervous symptoms have a resemblance to those of tabes dorsalis, and that they are dependent also upon an anatomical change in the posterior columns of the cord. Convulsions and psychical phenomena are also observed. A second form of chronic ergotism is called gangrenous ergotism. It results in dry gangrene of the hands and feet. A line of demarcation forms and the gangrenous parts slough off. The process may be attended by fever and pyæmia. The probable explanation is that the minute blood vessels become spasmodically contracted and thrombi form under the influence of the poison. The different symptoms are due in part to the different constituents of the ergot. Ergotoxin is prepared pure; in small doses it stimulates the smooth muscles; in large doses it paralyzes. Cornutin contains a convulsive poison.

36. **Poisonous Mushrooms.**—(1) *Poisoning from morels:* False morels ("morcheln" or "lorcheln") contain a poison which is readily soluble in hot water, and which decomposes if the morels be dried. Morels that have been dried or parboiled are therefore perfectly harmless, but their broth is poisonous. The symptoms are nausea, vomiting, diarrhea, headache, coma, and, above all, hemoglobinæmia and hemoglobinuria, associated with which is a hematogenous icterus. In severe cases death occurs, ushered in by convulsions. *Treatment* is symptomatic, and includes the administration of emetics, purgatives, and stimulants. (2) *Boletus satanas:* This is often confused with the edible mushroom or boletus. Choleriform gastro-enteritis, eventually with severe collapse. *Treatment:* Symptomatic. (3) *Poisoning from the toadstool* (*agaricus muscarius*): This contains the poisonous alkaloid muscarin, which causes diaphoresis, myosis, spasm of accommodation, salivation, bradycardia, and dyspnœa. It also contains muscaradin, which has an atropin-like

action ("fungous atropin"), and is usually present in toadstool intoxication. *Symptoms:* Excitement resembling drunkenness, delirium, tetanic and epileptiform convulsions, usually mydriasis, constriction in the throat, tachycardia, in severe cases sopor and death. *Treatment:* Evacuation of stomach and intestines. Where there is any muscarin effect, the physiological antidote, atropin, is used. Also morphin, chloral, stimulants. (4) *Poisoning from bulbous mushrooms (amanita phalloides)*, confused with young button mushrooms (champignons): After an incubation period of ten hours, digestive disturbances, later jaundice, somnolence, coma. The autopsy shows fatty degeneration of the liver, kidneys, and stomach, as in phosphorous poisoning.

37. **Indigenous Poisonous Plants.**—Hemlock (*conium maculatum*) has already been mentioned. Water hemlock (*cicuta virosa*) contains a powerful convulsive poison (*cicutoxin*). Golden rod (*cytisus laburnum*), not infrequently swallowed by children, gives the symptoms of cytisin poisoning—vomiting, paralytic conditions, dilated pupils. As a rule, recovery ensues. Related in their action are the alkaloids of belladonna (atropin, *vide supra*), of thorn apple (*datura stramonium*), and henbane (hyoscin). Monkshood (*aconitum napellus*) contains aconitin, and causes paræsthesiæ of the tongue and skin, vomiting, vertigo, syncope, severe cardiac collapse, paralysis of respiration. *Treatment:* Tannin, stimulants, artificial respiration. Meadow saffron (containing colchicin) causes gastro-enteritis (choleriform and dysenteriform), headache, vertigo, convulsions. *Treatment* is symptomatic. Milkweed (*euphorbia*) and crowfoot (*ranunculus*) are violent irritants to the gastrointestinal tract. Solanin is present in unripe, germinated, or decomposed potatoes, as well as in the berries of the potato plant (*solanum tuberosum*) and other solanum species (*S. nigrum*, nightshade; *S. dulcamara*, sweetwood, etc.). It induces gastro-enteritis, cardiac weakness, mydriasis. *Treatment:* Evacuation of gastrointestinal tract, stimulants.

38. **Poisoning from Meat and Sausages.**—This is due, on the one hand, to the meat of diseased animals—i. e., those infected with the bacillus enteritidis or paratyphosus B. (dysentery, puerperal processes of cows, septic polyarthritis of mammals and other septic affections), and, on the other hand, to meat infected postmortem. Regarding the latter, it may be said that ordinary decomposition is not harmful, and that only certain putrescent processes form poisonous products. On the other hand, meat (or sausages) may be the culture medium for toxin-producing bacteria, such as the abovementioned bacillus enteridis and paratyphosus, as well as the anaërobic bacillus botulinus, and may thus become poisonous without noticeably changing its appearance. Clinically, we can differentiate four forms of meat and sausage poisoning, besides various mixed forms:

1. *Gastro-enteritis*, due to decomposed meat, to meat containing the toxins of paratyphoid and enteritis bacilli, or even the bacilli themselves. This gastro-enteritis may resemble cholera and dysentery, and usually runs its course in a few days with fever and prostration. Death occurs from exhaustion. *Treatment:* Calomel, bolus; otherwise symptomatic. Plenty of fluids.

2. *Typhoid:* From the bacilli of the paratyphoid group, whereby there occurs not merely a local infection of the intestine, as in the preceding form, but also a general infection. Presents the clinical symptoms of abdominal

typhoid. *Differences:* Acute onset, also with chills, usually more marked roseola. Begins with enteritis, etc. This form may associate itself with an acute gastro-enteritis. *Treatment:* Symptomatic.

3. *Ptomatropinism:* From putrescent meat or meat from animals with suppurative processes. Characterized by various atropin symptoms (ptomatropin), as mydriasis, paralysis of accommodation, dryness of the mucous membranes, cessation of salivary secretion, dysphagia, tachycardia, vertigo, and the like; also intestinal paralysis, but usually gastro-enteritis. The phenomena last only a few days; the ocular symptoms, too, very soon disappear completely. *Treatment:* Castor oil, irrigation, ingestion of nourishment and liquids (if necessary, stomach tube).

4. *Botulism:* Due to the toxin of the anaërobic bacillus botulinus, which causes degeneration of the bulbar nuclei. This bacillus occasionally flourishes in thick sausages, in large pieces of meat, in preserves—i. e., wherever there is a lack of oxygen. The infected meat may have an almost entirely normal appearance and taste. *Symptoms:* Paresis of accommodation, mydriasis, immobility of the pupils, and paralysis of the ocular muscles (most frequently ptosis), dryness, absence of saliva, dysphagia, aphagia, aphonia, bradypnœa, severest constipation (intestinal paralysis), more rarely paralysis of the facial and hypoglossus nerves; also paralysis of the diaphragm, more often a great muscular weakness. Cerebral disturbances, disturbances of sensibility, convulsions, and atrophy of the muscles do not pertain to this picture. Death by paralysis of respiration or marasmus. Convalescence requires a long time, the visual disturbances persist several months. *Treatment:* Animal charcoal, evacuation of stomach and intestines (no calomel), botulismus antitoxin (diphtheria antitoxin is also effective); if necessary, artificial respiration.

39. **Fish Poisoning** (three forms):

1. *Choleriform ichthysmus:* Gastro-enteritis with collapse. Occurs from idiosyncrasy, from decayed fish, from preserves, from paratyphoid infections, from barb roe.

2. *Exanthematic ichthysmus:* Gastro-enteritis with urticaria, roseola, etc. Seen in idiosyncrasies from decayed fish.

3. *Neurotic ichthysmus* (ptomatropinism): Atropin symptoms as in type 3 of meat poisoning. Rapid course. Due to dried fish (sturgeon and others), especially in Russia (the Volga region).

Genuine botulism from canned fish also occurs. *Treatment:* Same as for meat poisoning.

40. **Poisoning from Mussels** (*Mytilus edulis*).—This is also common. A drawing feeling in the neck, a blunt feeling in the teeth, crawling and burning in the arms and legs, dullness in the head, mental excitability, feeling as if everything were light, as if the patient must fly; in the later stages dilated, immobile pupils, difficult speech, paresis and ataxia of the muscles; also nausea, skin eruptions (urticaria), fall of temperature. In severe cases death may ensue in a few hours. At the autopsy we find severe enteritis, enlargement of the spleen, and often a peculiar speckling of the liver. The poison of mussels has been isolated, and it is called mytilotoxin. Moreover, many "intoxications" occur from edible shellfish, especially from oysters, because the animals in question have been in impure water infected with typhoid bacilli and the like.

41. **Poisoning from Cheese.**—Vomiting, colicky pains, diarrhea, vertigo, sense of thoracic oppression, headache, languor, and disturbances of vision. *Treatment:* As in other poisonings from decayed foods.

42. **Snake Venom.**—In Germany the chief poisoning is from the bite of the common adder. The secretion of its fangs contains a poisonous albuminous body which acts as a ferment and causes rapid coagulation of the blood and thrombosis. The integument swells, assumes a blackish color (hemorrhagic œdema). Lymphangitis and lymphadenitis follow. Vomiting, collapse, hematuria, severe nervous symptoms (delirium, convulsions). In severe cases, death in a few hours or days. In mild cases, slow convalescence. *Treatment:* Suction of the poison, cauterization, and excision of the wound. Constriction of the limb above the site. Subcutaneous injection of three- to five-per-cent potassium permanganate solution into the neighborhood of the wound. Injections of phenol and of strychnin have also been recommended. Alcohol internally. For poisoning from the tropical serpents active antitoxins have been prepared.

APPENDIX III

FORMULÆ. BATHS AND HEALTH RESORTS

1. *Antipyretics*

1. Quininæ sulphatis, gr. xv–xxx (gm. 1–2).
Acidi sulphuric. dilut., q. s. ad solut.
Aq. menthæ pip., ℥ss. (15 c.c.).
M. S. To be taken at one dose.

(Antipyretic. In malaria, trigeminal neuralgia, etc.)

2. Quininæ hydrochlor., gr. vijss.–xv (gm. 0.5–1).
D. tal. pulv., No. 6.

S. Take one or two powders in a wafer.

3. Quininæ hydrochlor., gr. xxijss. (gm. 1.5).
Pulv. fol. digitalis, gr. vijss. (gm. 0.5).
Extract. glycyrrhizæ, q. s. ad pil. No. 30.

S. Take one or two pills thrice daily.

4. Sodii salicylicat., ʒj–gr. lxxv (gm. 4–5).
D. tal. pulv. No. 4.

S. Take one or two powders daily in wine, black coffee, or a wafer.

5. Sodii salicylicat., ʒij–ʒijss. (gm. 8–10).
Aquæ menthæ, ℥j (30 c.c.).

M. S. Divide into two or three parts, and use in twenty-four hours.

6. Acidi salicylici cryst. pulv., gr. vijss. (gm. 0.5).
D. ad capsulus gelatinosas vel geloduratas tal. pulv., No. 20.

S. Take one capsule every hour.

(In acute articular rheumatism.)

7. Salol., gr. xv–xxx (gm. 1–2).
D. tal. pulv., No. 10.

S. Take three or four powders daily in wafers.

8. Antipyrini, gr. xv–xxx (gm. 1–2).
D. tal. pulv., No. 10.
S. Take one or two powders in wafers, wine, or water.

9. Antifebrini, gr. iv–vijss. (gm. 0.25–0.5).
D. tal. pulv., No. 10.
S. Take one or two powders in capsules or in wine.

10. Salipyrini (Antipyrin. salicylat.) gr. xv–xxx (gm. 1–2).
D. tal. pulv., No. 10.
S. Take one or two powders in wafers or in wine.

11. Phenacetini, gr. vijss.–xv (gm. 0.5–1).
D. tal. pulv., No. 6.
S. To be taken in wine or wafers.

12. Pyramidoni, gr. iij–vijss. (gm. 0.2–0.5).
D. tal. pulv., No. 10.
S. Take two or three powders daily in wafers, seltzer, etc.

13. Antipyrini, gr. xv (gm. 1).
Phenacetini, gr. vijss. (gm. 0.5).
M. f. pulv. D. tal. pulv., No. 10.
S. Take one or two powders daily.
(For headaches, neuralgic pains, etc.)

14. Aspirini, gr. vijss.–xv (gm. 0.5–1).
D. tal. dos., No. 10 (or in tablets).
S. Take three to five powders (or tablets) daily.
(For articular rheumatism, etc.)

2. *Acids*

15. Acidi phosphorici, ʒss. (2 c.c.).
Aq. destillat., ℥iv (120 c.c.).
Syr. rubi idæi, ʒv (20 c.c.).
M. S. One tablespoonful every two hours.

16. Acid. hydrochlorici dil., ʒss. (2 c.c.).
Aq. destillat., ℥iv (120 c.c.).
Syr. rubi idæi, ℥ss. (15 c.c.).
M. S. One tablespoonful every two hours.

17. Acidi sulphurici dilut., ʒss. (2 c.c.).
Aquæ destillat., ℥iv (120 c.c.).
Syr. rubi idæi, ʒv (20 c.c.).
M. S. One tablespoonful every two hours.

18. Potassii carbon., ʒj (gm. 4).
Succi. citri recent. express., q. s. ad saturationem.
Aquæ, ℥iij (100 c.c.).
Syr. simpl., ʒv (20 c.c.).
M. S. One tablespoonful every hour or two.
(Potio Riveri.)

3. *Expectorants*

19. Ipecacuanhæ, gr. v–vijss. (gm. 0.3–0.5). Infunde.
Aq. fervidæ, ℥iv (120 c.c.). Adde.
Syr. althææ, ʒijss. (10 c.c.).
(May add morphini hydrochlor., gr. ss.–j [gm. 0.03–0.06].)
M. S. One tablespoonful every two hours.

20. Inf. ipecac., 0.3–0.5: 120 c.c.
Aq. amygdal. amar., ♏ xlv (3 c.c.).
Or extr. opii, gr. iij (gm. 2).
Extract glycyrrhizæ, ʒj (5 c.c.).
M. S. One tablespoonful every two hours.

21. Infus. senegæ, 10:150 c.c.
Ammonii chloridi, gr. xlv (gm. 3).
Extract. glycyrrhizæ, ʒijss. (10 c.c.).
M. S. One tablespoonful every hour or two.

22. Infus. senegæ, 10:150 c.c.
Liq. ammonii anisat., ʒj (4 c.c.).
Syr. simpl., ʒv (20 c.c.).
M. S. One tablespoonful every two hours.

23. Decoct. quillajæ, 5:180 c.c.
Syr. simpl., ʒijss. (10 c.c.).
M. S. One tablespoonful every two hours.
(Is said to be preferable to senega root.)

24. Pulv. ipecac., gr. ss. (gm. 0.03).
Opii, gr. $\frac{1}{8}$ (gm. 0.01).
Sacchari albi, gr. iij (gm. 0.2).
M. fiat pulvis. D. tal. pulv., No. 20.
S. Take one powder every hour or two.

25. Liq. ammonii anisat,
Tinct. stramonii,
Tinct. opii, ana ʒj (5 c.c.).
M. D. S. Fifteen to twenty drops in water thrice daily.
(In chronic bronchitis and emphysema.)

26. Liq. ammonii anisat., ʒj (5 c.c.).
Aq. amygdal. amar., ʒijss. (10 c.c.).
M. S. Twenty drops in water thrice daily.

27. Ammonii chloridi,
Extract. glycyrrhizæ, ana gr. xlv–ʒj (gm. 3–5).
Aqua fœniculi, ℥vj (180 c.c.).
M. S. One tablespoonful every two hours.
(Mistura solvens.)

28. Ammon. chloridi, ʒj (gm. 5).
Antimonii et potassii tartrat., gr. j (gm. 0.05).
Aq. fœniculi, ℥v (150 c.c.).
Extract. glycyrrhizæ, ʒijss. (10 c.c.).
M. S. One tablespoonful every two hours.

29. Antimonii pentasulphid., gr. ss. (gm. 0.03).
Ammonii chloridi.
Sacchari albi, ana gr. v. (gm. 0.3).
M. f. pulv. D. tal., No. 15.
S. Take one powder every three hours.

30. Antimonii pentasulphid., gr. j (gm. 0.05).
Morphin. hydrochlor., gr. $\frac{1}{12}$–$\frac{1}{6}$ (gm. 0.005–0.01).
Sacchari albi, gr. vijss. (gm. 0.5).
M. f. pulv. D. tal. pulv., No. 10.
S. Take one powder two or three times a day.

31. Apomorphin. hydrochlor., gr. ss.–j (gm. 0.03–0.05).
Acid. hydrochlor. dilut., ♏ viij (0.5 c.c.).
Morphin. hydrochlor., gr. ss. (gm. 0.03).
Aq. fœniculi, ℥v (150 c.c.).
M. D. in vitro nigro.
S. One tablespoonful every three hours.

32. Apomorphin. hydrochlor., gr. j (gm. 0.05).
Extract. glycyrrhizæ, q. s. ad pilulas, No. 50.
S. Take one to three pills every hour or two.

33. Acidi benzoici, gr. ij–v (gm. 0.1–3).
Sacchari albi, gr. vijss. (gm. 0.5).
M. f. pulvis. D. tal. pulv. ad chartam ceratam, No. 10.

S. Take one powder every hour or two.

34. Balsami peruviani, ʒj (5 c.c.).
Acaciæ, gr. xl (gm. 2.5).
Aqua destillat., ℥v (150 c.c.).
Fiat emulsio, cui adde
Syr. cinnamomi, ʒv (20 c.c.).

M. S. One tablespoonful every hour or two. Shake.

Balsam of Peru, balsam of copaiba, turpentine, myrtol, etc., are best given internally (e. g., in bronchorrhea and fetid bronchitis) in gelatin capsules.

3a. *Expectorants for Children*

35. Infus. ipecac., 0.1–0.3 : 100.
Aq. amygdal. amar., ♏ xxv (1.5 c.c.).
Syr. althææ, ℥ss. (15 c.c.).

M. S. One teaspoonful every two hours.

36. Infus. senegæ, 5 : 100.
Liq. ammonii anisat., ♏ xv (gm. 1).

M. S. One teaspoonful every two hours.

37. Ammonii et potassii tartrat., gr. ss. (gm. 0.03).
Aq. destillat., ℥iijss. (100 c.c.)
Syr. althææ, ʒv (20 c.c.).

M. S. One teaspoonful every two hours.

38. Apomorphin. hydrochlor., gr. ⅔ (gm. 0.04).
Aqua destillat., ℥ss. (15 c.c.).
Syr. althææ, ʒv (20 c.c.).

M. S. Take fifteen to twenty drops every hour.

39. Acidi benzoici,
Camphoræ, ana gr. ss–j (gm. 0.03–0.05).
Sacchari lactis, gr. v (gm. 0.3).
M. f. pulv. D. tal. pulv., No. 10, ad chartam ceratam.

S. Take one powder every two hours.

4. *Remedies for Inhalation*

40. Remedies for inhalation are employed in the following concentrations:
Sodium chlorid, 2–10 : 500.
Sodium bicarbonate, 2–10 : 500.
Ammonium chlorid, 2–5 : 500.
In addition, the mineral waters of Ems, Salzbrunn, etc., are often employed in inhalations.
Alum, 2–5 : 500.
Tannic acid, 2.5–10 : 500.
(Turpentine and Peruvian balsam, the latter diluted with two parts of alcohol, are poured in teaspoonful amounts upon hot water and the fumes then inhaled. The so-called turpentine pipe, which can also be used for the inhalation of other resinous remedies, is very serviceable. Carbolic-acid inhalations are best carried out with the aid of the Curschmann mask (*vide* Vol. I, page 205).[1]
Aq. amygdalarum amar., 5–10 : 500.
Tinct. opii, 1–3 : 500.
Ext. opii, 0.3–0.5 : 500.
Potassium bromid, 3–10 : 500.

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[1] For narcotics and cocain, *vide infra.*

5. *Other Prescriptions and Dosages for the Treatment of Diseases of the Organs of Respiration*

41. Acid. carbol,
Alcohol, ana ʒijss. (10 c.c.).
Aqua ammoniæ, ʒj (5 c.c.).
M. S. For inhalation.

42. The solutions most often used for nasal douches are:
Water, chamomile tea, sage tea.
Potassium chlorid, 15–50 : 1,000.
Sodium carbonate, alum, and tannin, 10–20 : 1,000.
Boric acid, salicylic acid, and carbolic acid, 5–10 : 1,000.
Potassium permanganate, 1–5 : 1,000.

43. The following solutions are best for application to the mucous membrane of the pharynx, nose, and larynx:
Silver nitrate, 1–15 : 50, aq. dest.
Alum and tannic acid, 5 : 25, aq. dest.
Tincture of iodin, plain or diluted with alcohol.
Borax, 5, with glycerin, 25.
Pure alcohol.
Iodized glycerin:
Iodii, gr. vijss. (gm. 0.5).
Potassii iodid., gr. iv (gm. 2.5).
Glycerini, ʒvj (25 c.c.).
M. S. External use.

44. Potassii vel sodii iodid., ʒj–ʒijss. (gm. 5–10).
Aq. destillat., ℥vij (200 c.c.).
M. S. Three or four teaspoonfuls to be taken daily in water.

(For dry bronchitis, fibrinous bronchitis, and particularly for bronchial asthma.)

45. Extract. belladonnæ, gr. $\frac{1}{6}$–$\frac{1}{3}$ (gm. 0.01–0.02).
Sacchari albi, gr. v (gm. 0.3).
M. f. pulv. D. tal. pulv., No. 15.
S. Take one powder thrice daily.
(For pertussis.)

46. Potassii bromid., ʒss.–j (gm. 2–5).
Aq. destillat., ℥iijss. (100 c.c.).
Syr. simpl., ʒv (20 c.c.).
M. S. One teaspoonful every three hours.
(For pertussis.)

47. Chloroformi, ℥j (30 c.c.).
Ætheris, ℥ij (60 c.c.).
Ol. terebinthæ, ʒijss. (10 c.c.).
M. S. Pour one or two teaspoonfuls on a handkerchief and inhale.
(For pertussis.)

48. Bromoformi, ♏ xlv (3 c.c.).
D. in vitro nigro.
S. Three to five drops in sweetened water thrice daily.
(For pertussis.)

49. Guaiacol. carbonat., gr. vijss. (gm. 0.5).
D. tal. pulv., No. 30.
S. Three powders daily.
(Chronic tuberculosis.)

50. Pyrenol, ʒj (gm. 4).
Aq. destillat., ℥iv (130 c.c.).
Syr. rubi idæi, ʒv (20 c.c.).
M. S. Three or four tablespoonfuls daily.
(In pneumonia, bronchitis, pertussis, etc.)

6. *Prescriptions for the Treatment of Diseases of the Mouth and Pharynx*[1]

51. Potassii chlorat., ʒijss. (gm. 10).
Aq. destillat., ℥x (300 c.c.).
M. S. Gargle, plain or diluted with equal parts of water.

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[1] For solutions for local application see also No. 43.

52. Aqua hydrogen. dioxid., ℥iijss. (100 c.c.) of a three-per-cent solution.
D. S. For gargling and mouth wash.
(Peroxid-of-hydrogen solution.)

53. Sodii borat., ʒijss. (gm. 10).
Aq. destillat., ℥x (300 c.c.).
M. S. Gargle.

54. Potassii chlorat., gr. xlv–lxxv (gm. 3–5).
Aq. destillat., ℥v (150 c.c.).
(With children, ad. syr. simpl., ʒijss. [10 c.c.].)
M. S. Slowly swallow one half teaspoonful every quarter to half hour.

(For ordinary sore throat, necrotic sore throat, and diphtheria.)

55. Hydrargyri chloridi corrosivi, gr. ⅓–j (gm. 0.02–0.05).
Aq. destillat., ℥ij (50 c.c.).
M. S. Swab the throat from four to six times daily.
(For beginning diphtheria.)

7. *Remedies for Regulating the Heart's Action*

56. Fol. digitalis, gr. xv–xxx (gm. 1–2).
Aq. fervidæ, ℥v (150 c.c.).
M. et fiat infusio.
S. One tablespoonful every two hours.

57. Fol. digitalis, gr. ij–iij (gm. 0.1–0.2).
D. tal. pulv. (ad. caps. amylac.), No. 20.
S. Take one powder three to five times daily.

58. Infus. digitalis, 1.5 : 120.
Liq. potass. acetici ʒj (30 c.c.).
Oxymel scillæ, ℥ss. (15 c.c.).
M. S. One tablespoonful every two hours.

59. Fol. digitalis, gr. ij (gm. 0.1).
Diuretini, gr. xv (gm. 1).
M. f. pulvis. D. tal. pulv., No. 10.
S. Take three powders daily.

60. Caffeinæ sodio-salicyl., gr. iij–v (gm. 0.2–0.3).
D. ad. capsul. amyl. tal. dos., No. 12.
S. Take four to six powders daily.

61. Fol. digitalis, gr. ij (gm. 0.1).
Caffeinæ sodio-salicyl., gr. iij (gm. 0.2).
M. f. pulv. D. tal. pulv., No. 12.
S. Take one powder thrice daily.

62. Digalen, ℥ss. (15 c.c.).
S. Fifteen drops thrice daily.

63. Tinct. strophanthi, ʒijss. (10 c.c.).
S. Ten to fifteen drops several times daily.

(Good stimulant in chronic, and particularly in acute, heart failure.)

64. Spiritus nitroglycerini, ♏ xv (1 c.c.).
Aq. destillat., ʒijss. (10 c.c.).
S. Ten to fifteen drops (increasing cautiously).
(For angina pectoris.)

8. *Excitants* (*analeptics*)

65. Camphoræ, gr. xxxvijss. (gm. 2.5).
Ol. amygdalorum, ʒijss. (10 c.c.).
M. S. For subcutaneous injection. An entire Pravaz syringeful (♏ xv [1 c.c.]) at once, repeated, if necessary.

66. Camphoræ, gr. ij–iijss. (gm. 0.1–0.15).

Sacchari lactis, gr. vjss. (gm. 0.4).

M. f. pulv. D. tal. dos. ad chartam ceratam, No. 10.

S. One powder every half hour.

67. Æther., ʒv (20 c.c.).

S. For subcutaneous injection; an entire syringeful [♏ xv] at once.

(Acetic ether and spirit of ether may be used in the same manner.)

68. Æther. sulphur. vel. acetici, ʒijss. (10 c.c.).

S. Ten to twenty drops on sugar.

(Spirit of ether may be used in like manner.)

69. Liq. ammonii anisat., ʒijss. (10 c.c.).

Spirit. æther.,

Tinct. aromat., ana ʒj (5 c.c.).

M. S. Twenty drops to be taken on sugar or in water.

70. Moschi, gr. j–iij (gm. 0.1–0.2).

Sacchari lactis, gr. vij (gm. 0.4).

M. f. pulvis. D. tal. dos. ad chartam cerat., No. 5.

S. One powder to be taken every two or three hours.

71. Alcohol. dilut.,

Tinct. aromat., ana ♏ xlv (3 c.c.).

Aq. destillat., ℥iv (120 c.c.).

Syr. simpl., ℥ss. (15 c.c.).

M. S. One tablespoonful every two hours.

(May be used instead of wine with poor patients.)

9. *Stomachics (digestives) and Bitters*

72. Acid. hydrochlor. dilut., ʒijss. (10 c.c.).

S. Ten to twenty drops in water half an hour after meals.

73. Pepsini, gr. vijss. (gm. 0.5).

D. tal. pulv., No. 10.

S. Take one powder in water after meals.

74. Tinct. cinchonæ compos., ʒvj (25 c.c.).

Acid. muriat. dilut., ʒj (4 c.c.).

Pepsini, ʒss. (gm. 2).

M. S. Twenty to thirty drops in water after meals.

75. Tinct. amaræ, ʒijss. (10 c.c.).

Tinct. aromat., ʒj (5 c.c.).

M. S. Fifteen to twenty drops.

76. Tinct. gentianæ, ʒijss. (10 c.c.).

Tinct. aromat.,

Syr. aurantii, ana ʒj (5 c.c.).

M. S. Twenty to thirty drops.

77. Tinct. cinchonæ compos.,

Tinct. aurantii, ana ℥ss. (15 c.c.).

M. S. Twenty to twenty-five drops.

78. Tinct. nucis vomicæ.,

Aq. laurocerasi, ana ʒijss. (10 c.c.).

M. S. Ten to fifteen drops twice or thrice daily.

79. Orexini tannici, gr. v–vijss. (gm. 0.3–0.5).

D. tal. pulv., No. 10.

S. One or two powders daily in wafers.

(Is said to stimulate the appetite.)

80. Bismuthi subnitrat., gr. v (gm. 0.3).
Sacchari albi, gr. iij (gm. 0.2).
M. f. pulv. D. tal. pulv., No. 15.
S. Three or four powders daily.

81. Bismuthi subnitrat., gr. v (gm. 0.3).
Morphinæ hydrochlorid., gr. ⅙ (gm. 0.01).
Sacchari albi, gr. iij (gm. 0.2).
M. f. pulv. D. tal. pulv., No. 10.
S. Two or three powders daily.
(For cardialgia.)

82. Bismuthi subcarbonat., ʒss.–j (gm. 2–5).
Sodii bicarbonat., ℥j (gm. 30).
Elæosacchari menthæ, ʒj (gm. 5).
M. f. pulv. D. ad scatulam.
S. Take a knife-pointful thrice daily.

83. Magnesiæ carbonat., ʒv (gm. 20).
Elæosacchari menthæ, ʒijss. (gm. 10).
M. f. pulv.
S. Take a knife-pointful twice or thrice daily.
(For heartburn.)

84. Sodii bicarbonat., ℥j (gm. 30).
Elæosacchari menthæ, ʒijss. (gm. 10).
M. f. pulv.
S. Knife-pointful.

85 Resorcini, ʒss. (gm. 2).
Aqua destillat., ℥vj (180 c.c.).
Syr. simpl., ʒv (20 c.c.).
M. D. in vitro nigr.
S. One tablespoonful every two hours.

86. Decoct. condurango, 15:150.
Syr. aurantii, ℥ss. (15 c.c.).
M. S. One tablespoonful every two or three hours.
(Stomachic, particularly in cancer of the stomach.)

87. Fluidext. condurango, ℥iv (120 c.c.).
Syr. aurantii, ℥j (30 c.c.).
M. S. One half to one teaspoonful thrice daily.

10. *Emetics*

88. Ipecacuanhæ, gr. xv (gm. 1).
Ammonii et potassii tartrat., gr. j (gm. 0.05).
M. f. pulvis. D. tal. dos., No. 4.
S. Take one powder every fifteen minutes until they act.

89. Ipecacuanhæ, gr. xv–xxx (gm. 1–2).
Ammonii et potassii tartrat., gr. ss.–j (gm. 0.03–0.05).
Aq. destillat., ℥j (30 c.c.).
Oxymel scillæ., ℥ss (15 c.c.).
M. S. A teaspoonful every ten minutes until the action desired is obtained.

90. Apomorphini hydrochloridi, gr. ⅔ (gm. 0.04).
Aqua destillat., ʒss. (2 c.c.).
M. S. Inject ♏ x–xv (0.6–1 c.c.).

91. Cupri sulphat., gr. xv (gm. 1).
Aqua destillat., ℥jss. (50 c.c.).
M. S. ʒj repeated until desired action is obtained.

11. *Laxatives* (*purgatives*)

92. Olei ricini, ℥ij (60 c.c.).
S. One tablespoonful every hour or two.

93. Olei ricini, ℥j (30 c.c.).
Acaciæ, gr. xv (gm. 1).
Fiat emulsio cum.
Aq. destillat., ℥ijss. (75 c.c.).
Syr. amygdal., ℥ss. (15 c.c.).
M. S. Teaspoonful every one or two hours.

94. Hydrargyri chlorid. mit., gr. v–vijss. (gm. 0.3–0.5).
D. tal. pulv. ad capsul. amyl., No. 5.
S. Take one powder every three hours until they act.

95. Sodii sulphat., ℥jss. (gm. 50).
Sodii bicarbonat., ʒjss. (gm. 6).
Sodii chlorati, gr. xlv (gm. 3).
M. f. pulvis.
S. One or two teaspoonfuls in a cup of warm water every morning.

96. Magnesii sulphat., ℥ij (gm. 60).
Sodii bicarbonat., ʒiij (gm. 12).
M. f. pulv.
S. One teaspoonful in a cup of water.

97. Sennæ, ℥ss. (gm. 15).
Anisi, ʒj (gm. 5).
Aq. fervidæ, ℥v (150 c.c.).
M. et fiat infusio; adde
Sodii sulphat., ʒv (gm. 20).
M. S. Tablespoonful hourly.

98. Frangulæ, ℥ijss. (gm. 80).
Sennæ,
Menthæ pip., ana ʒv (gm. 20).
M. et fiat species. A teaspoonful to a cup of tea. Take at night.

99. Pulv. rhei, ʒj (gm. 5).
Sodii vel magnesii sulphat., ℥ss. (gm. 15).
Zingiberis, ʒss. (gm. 2).
M. et fiat pulvis.
S. One half to one teaspoonful at a dose.

100. Rhei.
Sulphur. præcip.,
Potassii bitartrat.,
Anisi, ana ʒj (gm. 5).
Sacchari albi, ʒv (gm. 20).
M. et fiat pulv.
S. Knife-pointful.
(In hemorrhoids.)

101. Rhei.
Extr. rhei compos., ana gr. xx (gm. 1.5).
Extr. belladonnæ, gr. ij (gm 0.1).
M. et fiant pilulæ, No. 30.
S. Two to four pills daily.

102. Ext. aloes.
Sapon. jalapini, ana gr. xxi (gm. 1.5).
M. et fiant pilulæ, No. 30.
S. Two to four pills daily.

103. Extr. aloes,
Rhei., ana gr. xv (gm. 1).
Extr. colocynthidis,
Cambogiæ, ana gr. vijss. (gm 0.5).
M. et fiant pilul., No. 30.
S. Two or three pills daily.

104. Extr. aloes, gr. vijss. (gm. 0.5
Extr. rhei. comp., gr. xlv (gm 3).
Ferri, gr. xlv (gm. 3).
M. et fiant pil., No. 100.
S. One or two pills at a dose.

105. Extr. aloes, gr. xlv (gm. 3).
Extr. belladonnæ, gr. vijs (gm. 0.5).
Saponis, q. s. ad pilulas, No. 30
S. One or two pills at a dose.

106. Podophyllini, gr. ss.–v (gm 0.03–0.3).
Sacchari albi, gr. v (gm. 0.3).
M. et fiat pulvis. D. tal. pulv. No. 10.
S. One or two powders daily a required.

107. Decoct. tamarind., 50 : 250.
Magnesii sulphat., ʒvj (gm 25).
M. S. Drink one half to on glass every morning.

108. Fluidext. cascaræ sagradæ, ℥j (gm. 30) [or fluidext. cascaræ sagrad. aromatic.].

S. One half to one teaspoonful once or twice daily.

109. Glycerini, ♏ xlv–ʒj (3–5 c.c.).

S. To be injected with a small syringe into the rectum.

(Also glycerin suppositories.)

12. *Astringents and Antidiarrheal Remedies*

110. Acidi tannici, gr. j–ij (gm. 0.05–0.1).
Opii, gr. ⅓ (gm. 0.2).
Sacchari, gr. j (gm. 0.5).
M. et f. pulv. D. tal. pulv., No. 10.

S. One powder every two or three hours.

111. Tannigen or tannalbin, gr. vijss.–xv (gm. 0.5–1).
D. tal. pulv., No. 10.

S. One powder three times a day.

112. Argenti nitrat., gr. ij–iij (gm. 0.1–0.2).
Pulv. opii, gr. xxx (gm. 2).
Gentian. et extract. gentian., q. s. ad pilulas, No. 50.

S. Four to six pills daily.

(Chronic diarrhea.)

113. Argenti nitrat., gr. ij (gm. 0.1).
Aq. destill., ℥jss. (50 c.c.).
Glycerini, ʒijss. (10 c.c.).
M. D. in vitro nigro.

S. One teaspoonful every two hours.

(For diarrhea of children.)

114. Bismuthi subnitrat., gr. vijss.–xv (gm. 0.5–1).
Opii, gr. ⅓ (gm. 0.02).
Sacchari albi, gr. v (gm. 0.3).
M. et f. pulv. D. tal. pulv., No. 12.

S. Two or three powders daily.

(Chronic intestinal ulcers.)

115. Bismuthi subnitrat., ʒj (gm. 5).
Mucilag. acaciæ,
Syrup. simpl., ana ℥ss. (15 c.c.).
Aq. destillat., ℥iv (120 c.c.).

M. S. One tablespoonful every hour or two. Shake.

(For dysentery, etc.)

116. Bismuthi salicylat., gr. ij–v (gm. 0.1–0.3).
Pulv. gummos., gr. vijss. (gm. 0.5).
M. et fiat pulvis. D. tal. pulv., No. 12.

S. One powder every two hours.

(For chronic diarrhea, intestinal catarrh of children.)

117. Plumbi acetat., gr. ss.–j (gm. 0.03–0.05).
Opii, gr. ⅙–⅓ (gm. 0.01–0.02).
Sacchari albi, gr. vijss. (gm. 0.5).
M. et ft. pulv. D. tal. pulv., No. 12.

S. One powder every two or three hours.

118. Decoct. hæmatoxyli, 5–10 : 150.
Syr. simpl., ℥j (30 c.c.).

M. S. One tablespoonful every two hours.

119. Infus. calumbæ, 15 : 150.
Elixir aurantii, ʒj (5 c.c.).
Syr. simpl., ʒvj (25 c.c.).

M. S. Tablespoonful every two hours.

120. Decoct. krameriæ, 5–10 : 120.
Tinct. aromat., ʒj (5 c.c.).
Syr. simpl., ℥ss. (15 c.c.).
M. S. Tablespoonful every two hours.

121. Mucilag. acaciæ,
Syr. simpl., ana ʒv (20 c.c.).
Aq. destillat., ℥iv (120 c.c.).
Tinct. opii, ♏ xv–xxx (1–2 c.c.).
M. S. Tablespoonful every two hours.

122. Ol. amygdal., ʒijss. (10 c.c.).
Acaciæ, ʒj (gm. 5).
Aq. destillat., ℥iv (120 c.c.).
Fiat emulsio, adde
Syr. simpl., ʒv (20 c.c.).
May add tinct. opii, ♏ xxx (2 c.c.).
M. S. Tablespoonful every two hours.

123. Naphthaleni, gr. iij–vijss. (gm. 0.2–0.5).
D. tal. pulv., No. 10.
S. Three or four powders daily in wafers.

124. Decoct. salipis, 1 : 200.
Vini opii, gtt. iij–v.
D. S. One teaspoonful every one to two hours.

125. Hydrargyri chlorid. mit., gr. $\frac{1}{6}$ (gm. 0.01).
Opii, gr. $\frac{1}{20}$–$\frac{1}{12}$ (gm. 0.003–0.005).
Sacchari lactis, gr. v (gm. 0.3).
M. et ft. pulv. D. tal. pulv., No. 12.
S. One powder every two or three hours.
(In diarrhea of children.)

126. Creosoti, ♏ ij–iv.
Aq. destillat., ℥j (35 c.c.).
Syr. althææ, ℥ss. (15 c.c.).
M. S. Teaspoonful every two hours.
(In diarrhea of children.)

13. *Anthelmintica*

127. **Trochisci santonini, ana gr. j (gm. 0.05), No. 10.**
S. Two troches morning and night.
(In ascarides and oxyuris.)

128. Santonicæ, ʒj (gm. 5).
Jalapæ, gr. xv (gm. 1).
Syrupi, ʒvj (25 c.c.).
M. et fiat electuarium.
S. Take in three doses.

129. Extract. filicis, ʒij (gm. 8).
Muc. acaciæ, ℥j (30 c.c.).
Syr. simpl., ʒv (20 c.c.).
S. Take in two portions within half an hour.
(The simplest and best way to administer the extract of male fern is in gelatin capsules, each containing gr. xv–xxx [gm. 1–2] of the drug.)

130. Pelletierinæ tannat., gr. v–vijss. (gm. 0.3–0.5).
S. Take in water, and follow in half an hour with a purge.
(Said to be effectual.)

14. *Diuretics*

131. Liquor potassii acetici,
Syr. simpl., ana ʒvj (25 c.c.).
Aq. fœniculi, ℥iijss. (100 c.c.).
S. Tablespoonful every two hours.

132. Aceti scillæ, ℥j (30 c.c.).
Potassii carbonat. q. s. ad perfectam saturationem.
Aq. petroselini, ℥iv (120 c.c.).
Syr. simpl., ʒv (20 c.c.).
M. S. One tablespoonful every two hours.

133. Sodii acetat., ʒijss (gm. 10).
Aq. petroselini, ℥v (150 c.c.).
M. S. One tablespoonful every two hours.

134. Infus. scillæ, 1.5 : 150.
Tartar. boraxat., ℥ss. (gm. 15).
Syr. simpl., ℥ss. (15 c.c.).
M. S. One tablespoonful every two hours.

135. Ononidis,
Lign. juniperi,
Fruct. juniperi,
Fruct. petroselini, ana ʒvj (gm. 25).
M. et f. species.
D. S. One teaspoonful infused as a tea.

136. Diuretini (theobrominæ sodio-salicylat.), gr. xv–xxx (gm. 1–2).
D. tal. pulv., No. 10.
S. Take two or three powders in wafers daily.

137. Sodio-theocini acetat., gr. v–vijss. (gm. 0.3–0.5).
D. tal. pulv., No. 12.
S. One powder twice or thrice daily.

138. Agurini, gr. vijss.–xv (gm. 0.5–1).
D. tal. pulv., No. 12.
S. Three or four powders daily in wafers.

139. Euphyllin, gr. ij (gm. 0.1).
D. in original tablets, No. 20.
S. Three to five tablets daily.
(Euphyllin may also be prescribed in suppository form.)

140. Hydrargyri chlorid. mit., gr. iij (gm. 0.2).
D. tal. pulv. in capsulis, No. 10.
S. Three powders daily.
(As a diuretic in heart affections; may be combined with digitalis.)

15. *Diaphoretics*

141. Pilocarpinæ hydrochlorat., gr. iij (gm. 0.2).
Aq. destillat., ʒijss. (10 c.c.).
M. D. ad. vitrum amplum.
S. Ten to fifteen minims for hypodermic injection.

142. Pilocarpinæ hydrochlorat., gr. v (gm. 0.3).
Ext. glycyrrhizæ, q. s. et fiant pilulæ, No. 30.
S. One or two pills.

143. Pulv. ipecac. comp., gr. v (gm. 0.3).
Sacchari albi, gr. iij (gm. 0.2).
M. et ft. pulv. D. tal. pulv., No. 10.
S. One powder while in a hot pack.

144. The following are regarded as diaphoretic teas:
Chamomile flowers.
Elder flowers.
Linden flowers.

16. *Narcotics and anæsthetics*

145. Morphinæ hydrochloridi, gr. $\frac{1}{6}$ (gm. 0.01).
Sacchari albi, gr. vijss. (gm. 0.5).
M. et ft. pulvis. D. tal. pulv., No. 10.
S. Morphin powder. Take one at night.

146. Morphinæ hydrochloridi, gr. j–ij (gm. 0.05–0.1).
Aq. amygdal. amar., ʒijss. (10 c.c.).
M. S. Morphin drops. Dose, fifteen to twenty.

147. Morphinæ hydrochlorid., gr. iv (gm. 0.25).

Aq. destillat., ʒvj (25 c.c.).

D. S. One-per-cent morphin solution for hypodermic injection. One Pravaz syringeful [♏ xv] equals gr. $\frac{1}{6}$ (gm. 0.01) of morphin.

148. Morphinæ hydrochlorid., gr. j (gm. 0.05).

Syr. simpl., ℥j (30 c.c.).

M. S. One half to one teaspoonful.

149. Codeinæ phosphat., gr. $\frac{1}{3}$ (gm. 0.02).

Sacchari albi, gr. vijss. (gm. 0.5).

M. et ft. pulv. D. tal. pulv., No. 10.

S. One or two powders daily.

150. Heroinæ hydrochlorid., gr. $\frac{1}{20}$–$\frac{1}{12}$ (gm. 0.003–0.005).

Sacchari albi, gr. vijss. (gm. 0.5).

M. et ft. pulv. D. tal. pulv., No. 10.

S. Two or three powders daily.

151. Chloral. hydrat., ʒss.–j (gm. 2–5).

Aq., destillat.,

Syr. aurantii, ana ʒj (5 c.c.).

M. S. Take in one or two doses.

152. Chloral. hydrat., gr. xl (gm. 2.5).

Aq. destillat., ʒijss. (10 c.c.).

M. S. To be taken at night in half a glass of milk.

153. Chloralamid., gr. xxx (gm. 2).

D. tal. pulv., No. 5.

S. One powder to be taken in a wafer at night.

154. Sulphonal, gr. xv–xxx (gm. 1–2).

D. tal. pulv., No. 6.

S. Take one powder in plenty of fluid two hours before retiring.

155. Trional, gr. xv–xxx (gm. 1–2).

D. tal. pulv., No. 6.

S. One powder to be taken in a wafer at night.

156. Veronal, gr. vijss.–xv (gm. 0.5–1).

D. tal. pulv., No. 6.

S. One powder in a cup of tea at night.

157. Potassii bromidi, gr. xxx-xlv (gm. 2–3).

D. tal. pulv., No. 5.

S. Take one powder dissolved in a glass of water at night.

158. Cannabini tannat., gr. v–vijss. (gm. 0.3–0.5).

Sacchari albi, gr. iij (gm. 0.2).

M. et ft. pulv. D. tal. pulv., No. 6.

S. One powder at night.

(Expensive!)

159. Extr. cannabis indicæ, gr. iij–xv (gm. 0.2–1).

Sacchari albi, gr. v (gm. 0.3).

M. et ft. pulv. D. tal. pulv., No. 6.

S. One powder at night.

160. Paraldehyd, ʒj–ʒjss. (4–6 c.c.).

D. S. Take in water.

(In delirium tremens. Restlessness of alcoholics.)

161. Dormiol, gr. vijss.–xv (gm. 0.5–xv).

D. tal. pulv., No. 6.

S. One powder in a wafer or water at night.

162. Cocainæ hydrochlorid., gr. vijss.–xv–xxijss. (0.5–1–1.5).
Aq. destillat., ʒij (8 c.c.).
Alcohol, ʒss. (2 c.c.).
M. S. Paint externally.

163. Anæsthesini, gr. v–vijss. (gm. 0.3–0.5).
D. tal. pulv., No. 10.
S. One powder twice or thrice daily.
(Cardialgia. Vomiting.)

164. Cocainæ hydrochlorid., gr. vijss. (gm. 0.5).
Spir. vini, q. s. ad. solut.
Aq. destillat., ʒijss. (10 c.c.).
D. S. For hypodermic injection.
Dose, one quarter to one half Pravaz syringeful (♏ iv–viij).
(All cocain solutions are more stable if, instead of pure water, a 1:10,000 solution of bichlorid of mercury be used.)

17. *Tonics*

165. Ferri reducti, gr. j–iij (gm. 0.05–0.2).
Sacchari lactis, gr. vijss. (gm. 0.5).
M. et ft. pulvis. D. tal. pulv., No. 20.
S. Two powders daily.

166. Ferri oxid. saccharat. solut., ℥j (gm. 30).
D. ad scatulam.
S. Knife-pointful thrice daily.

167. Ferri carbonat. saccharat., ℥j (gm. 30).
D. S. Knife-pointful thrice daily.

168. Ferri sulphat.,
Potassii carbonat., ana ʒij–ʒijss. (gm. 8–10).
Tragacanthæ, q. s. ad pilulas, No. 100.
S. Two to four pills thrice daily (Blaud's pills).

169. Ferri lactat.,
Succi liquir. dep. ana ʒjss. (gm. 6).
M. et fiant pilulas, No. 100.
S. Two or three pills thrice daily.

170. Ferri lactat.,
Ext. cinchonæ aquos., ana ʒjss. (gm. 6).
Muc. acaciæ, q. s. ad pilulas, No. 100.
S. Two or three pills thrice daily.

171. Tinct. ferri chlorati æthereæ,
Aq. cinnamomi, ana ʒvj (25 c.c.).
M. S. Thrice daily one half to one teaspoonful in wine.

172. Liquoris ferri albuminati, ℥ij (60 c.c.).
S. One teaspoonful thrice daily.

173. Quinin sulphat.,
Ferri sulphat., ana gr. xlv (gm. 3).
Glycyrrhizæ, q. s. ad pilulas, No. 60.
S. One or two pills thrice daily.

174. Syrup. ferri iodid,
Syrup. simpl., ana ℥j (30 c.c.).
M. S. One teaspoonful thrice daily.

175. Arseni trioxid., gr. v (gm. 0.3).
Piperis, gr. xlv (gm. 3).
Extr. glycyrrhizæ, q. s. ad pilulas, No. 100.
S. Two or three pills daily.

176. Ferri redacti, ʒj (gm. 5).
Acid. arsenios., gr. iij (gm. 0.2).
Extr. gentianæ, q. s. ad pilul., No. 100.
S. One or two pills twice or thrice daily.

18. *Nervines*

177. Potassii bromidi, gr. xv–xlv (gm. 1–3).
D. tal. pulv. ad chart. ceratam, No. 20.
S. Two or three powders in one half glass of water or seltzer daily.

178. Liquor. potassii arsenit. (= solut. Fowleri),
Aq. menthæ, ana ʒijss. (10 c.c.).
M. S. Ten drops thrice daily. Dose to be gradually increased.[1]

179. Argenti nitrat., gr. xv (gm. 1).
Argillæ, ʒijss. (gm. 10).
Aq. destillat. q. s. ut fiant pil., No. 100.
S. One or two pills thrice daily before meals.

180. Extr. ergotæ, ʒj (gm. 5).
Extr. glycyrrhizæ, q. s. ad pilulas, No. 100.
S. One to three pills thrice daily.

181. Atropinæ sulphat., gr. ⅙ (gm. 0.01).
Extr. glycyrrhizæ, q. s. ut fiant pilulæ, No. 20.
S. Two or three pills daily.

182. Scopolaminæ hydrobromidi, gr. ⅙ (gm. 0.01).
Aq. destillat., ʒijss. (10 c.c.).
S. Five to fifteen minims subcutaneously.
(In paralysis agitans, tremor, and conditions of excitement.)

183. Zinci oxidi, gr. ⅛–vijss. (gm. 0.01–0.5).
Sacchari albi, gr. v (gm. 0.3).
M. et ft. pulv. D. tal. pulv., No. 20.
S. Two or three powders daily.

184. Zinci oxidi, ʒj (gm. 5).
Extract. belladonnæ, ʒss. (gm. 2).
Extract. valerianæ, ʒj (gm. 5).
Pulv. rad. valerianæ, q. s. ut fiant pilul., No. 100.
S. One pill thrice daily.
(For Epilepsy, Chorea, etc.)

185. Zinci valerianat, gr. xv (gm. 1).
[Vel. extr. belladonnæ, gr. ij (gm. 0.1).]
Extr. gentianæ, q. s. ut fiant pil., No. 20.
S. Two pills twice daily.
(For neuralgias, etc.)

186. Auri et sodii chlorid., gr. iv (gm. 0.25).
Extr. glycyrrhizæ, q. s. ad pil., No. 50.
S. One pill thrice daily.

187. Aconitinæ, gr. j (gm. 0.05).
Extract. hyoscyami, gr. xv (gm. 1).
Extract. glycyrrhizæ, ʒj (gm. 4).
M. et fiant pilulæ, No. 50.
S. One or two pills twice a day.

188. Tinct. valerianæ æthereæ,
Aq. amygdal. amar., ana ʒijss. (10 c.c.).
M. S. Twenty to thirty drops thrice daily.

189. Strychninæ nitrat., gr. iij–vijss. (gm. 0.2–0.5).
Extr. glycyrrhizæ, q. s. ad pilulas, No. 100.
S. One pill thrice daily.

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[1] For other arsenic prescriptions, *vide* Nos. 175 and 176.

190. Ferri lactat., ʒj (gm. 4).
Extr. cinchonæ aquos., ʒss. (gm. 2).
Extr. nucis vomicæ, gr. xv (gm. 1).
Extr. glycyrrhizæ, q. s. ad pilul., No. 100.
D. S. Three to five pills daily.

191. Sodii nitrit., ʒij (gm. 2).
Aqua destillatæ, ℥iv (120 c.c.).
M. One tablespoonful three or four times daily.[1]
(For angina pectoris, asthma, migraine.)

19. *Liniments and Ointments*

192. Olei olivæ,
Chloroformi, ana ℥ij (60 c.c.).
M. S. To be rubbed in.

193. Spirit. saponati,
Chloroformi, ana ℥ij (60 c.c.).
M. S. To be rubbed in.

194. Spirit. camphoræ, ℥iij (90 c.c.).
Ol. juniperi, ℥ss. (15 c.c.).
M. S. To be rubbed in.

195. Potassii iodid., gr. xv–lxxv (gm. 1–5).
Vaselini, ʒvj (gm. 25).
M. fiat unguentum.
S. External use.

196. Iothion, gr. xlv (gm. 3).
Vaselini,
Lanolini, ana ʒijss. (gm. 10).
M. et fiat unguentum.
S. External use.

197. Iodoformi, gr. xv (gm. 1).
Collodii, ℥ss. (15 c.c.).
M. S. External use.

198. Balsami peruviani, gr. xv (gm. 1).
Unguent. simpl., ℥j (gm. 30).
M. et f. unguentum.
S. External use.
(For bedsores.)

199. Veratrinæ, gr. ij–vijss. (gm. 0.1–0.5).
Unguent. simpl., ʒijss. (gm. 10).
M. et fiat unguentum.
S. Veratrin ointment.
(Externally in neuralgias and the like.)

200. Unguent. hydrargyri dilut., gr. xxx–lxxv (gm. 2–5).
D. tal. dos. ad chartam cerat., No. 12.
S. To be rubbed in as directed.
(For inunctions in syphilis, etc.)

20. *A Summary of the Most Important Baths and Health Resorts*

1. *Indifferent Thermal Waters.*—Badenweiler, Gastein, Johannesbad, Leuk, Pfäffers, Ragatz, Schlangenbad, Teplitz, Warmbrunn, Wildbad, etc.

2. *Alkaline Waters.*—Ems, Neuenahr, Obersalzbrunnen, Vichy, etc.

3. *Alkaline-saline Waters.*—Carlsbad, Elster (Salzquelle), Franzensbad, Marienbad, Kissingen, Tarasp-Schuls, etc.

4. *Saline Baths.*—(*a*) Cold salines: Arnstadt, Aussee, Colberg, Frankenhausen, Gmunden, Hall, Ischl, Kösen, Kreuznach, Oeynhausen, Reichenhall, Salzungen, Sulza, Wittekind, etc. (*b*) Warm salines: Baden-Baden, Münster and Stein, Wiesbaden.

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[1] For prescriptions containing quinin, antipyrin, antifebrin, phenacetin, see Nos. 1, 8, 9, and 11.

5. *Sodium Chlorid Drinking Waters.*—Baden-Baden, Homburg, Kissingen, Wiesbaden, etc.

6. *Saline-carbonated Waters.*—(*a*) Cold: Homburg, Kissingen, Salzschlirf, Soden, etc. (*b*) Warm: Nauheim, Oeynhausen, and Soden.

7. *Iodized and Bromized Salines.*—Adelheidsquelle, Hall, Königsdorff-Jastrzemb, Kreuznach, Krankenheil-Tölz, etc.

8. *Sulphur Springs.*—Aix-la-Chapelle, Baden near Vienna, Baden near Zurich, Eilsen, Kreuth, Leuk, Neundorf, Weilbach, etc.

9. *Chalybeate Waters.*—Alexisbad, Brückenau, Cudowa, Driburg, Elster, Franzensbad, Liebenstein, St. Moritz, Pyrmont, Rippoldsau, Schwalbach, Spaa, Steben, etc.

10. *Sea Baths.*—(*a*) Baltic Sea: Schwarzort, Cranz, Zoppot, Colberg, Dievenow, Misdroy, Swinemünde, Heringsdorf, Zinnowitz, Rügen (Sassnitz, Lohme, Göhren, etc.), Doberan und Heiligendamm, Warnemünde, Travemünde, and many others. (*b*) North Sea: Wyk-auf-Föhr, Westerland-Sylt, Heligoland, Borkum, Spiekeroog, Wangeroog, Norderney, Ostend, Blankenberghe, Scheveningen, and many others.

11. *Iron-peat Baths.*—Cudowa, Elster, Franzensbad, Marienbad, Pyrmont, Reinerz, Schmiedeberg, etc.

12. *Mud Baths.*—Aix-la-Chapelle, Eilsen, Neundorf, Baden near Vienna, etc.

13. *Sand Baths.*—Blasewitz near Dresden, Köstritz, etc.

14. *Grape-cure Resorts.*—Arco, Botzen, Dürkheim, Gries, Meran, Montreux, Vevey, and many others.

APPENDIX IV

TABLE OF WEIGHTS AND MEASURES

TABLE OF RELATION OF U. S. FLUID TO METRIC MEASURE

Minims.		Cubic Centimeters.	Minims.		Cubic Centimeters.	Fluid Drachms.		Cubic Centimeters.	Fluid Ounces.		Cubic Centimeters.
1	=	.06	30	=	1.85	4	=	14.79	4	=	118.24
2	=	.12	40	=	2.46	6	=	22.18	6	=	177.39
5	=	.31							8	=	236.53
10	=	.62	Fluid Drachms.		Cubic Centimeters.	Fluid Ounces.		Cubic Centimeters.	12	=	354.82
15	=	.92	1	=	3.70	1	=	29.57	16	=	473.11
16¼	=	1.00	2	=	7.39	2	=	59.10			
20	=	1.23	3	=	11.09	3	=	88.67			

TABLE OF RELATION OF TROY WEIGHT TO GRAMS

Grains.		Grams.	Grains.		Grams.	Drachms.		Grams.	Ounces.		Grams.
⅛	=	.008	8	=	.52	1	=	3.89	1	=	31.1
⅙	=	.011	10	=	.65	1½	=	5.83	1½	=	46.6
¼	=	.016	15	=	.97	2	=	7.77	2	=	62.2
½	=	.032	15.43	=	1.00	3	=	11.66	3	=	93.3
1	=	.065	20	=	1.29	4	=	15.55	4	=	124.4
2	=	.13	30	=	1.94	6	=	23.3	6	=	186.6
4	=	.26	40	=	2.59				8	=	248.8
5	=	.32	61.73	=	4.00						
6	=	.39									

Table of Comparison between Centigrade and Fahrenheit Scales

Degrees Centigrade.		Degrees Fahrenheit.	Degrees Centigrade.		Degrees Fahrenheit.
25	=	77	36	=	96.8
26	=	78.8	37	=	98.6
27	=	80.6	38	=	100.4
28	=	82.4	39	=	102.2
29	=	84.2	40	=	104.
30	=	86	41	=	105.8
31	=	87.8	42	=	107.6
32	=	89.6	43	=	109.4
33	=	91.4	44	=	111.2
34	=	93.2	45	=	113
35	=	95			

The Metric System in Medicine

Old Style.		Metric.	
♏ j or gr. j	=		06 gm.
f ʒ j or ʒ j	=	4	"
f ℥ j or ℥ j	=	32	"

The decimal *line,* instead of *points,* makes errors impossible. As 0.06 (drug) is less than a grain, while 4 and 32 (vehicle) are more than the drachm and ounce, there is no danger of giving too large doses of strong drugs.

C.c. (cubic centimeters), used for gm. (grams), causes an error of five per cent (excess).

A teaspoonful is usually 5 grams; a tablespoonful, 20 grams.

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